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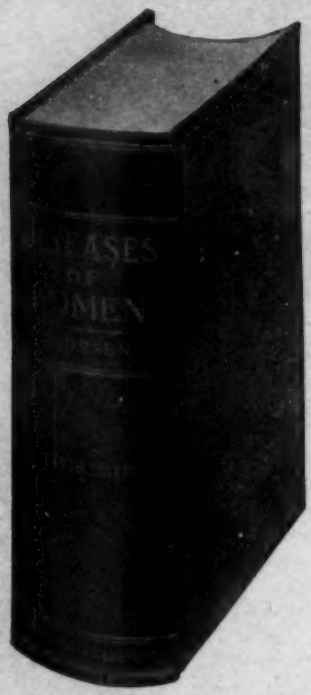

# Diseases of Women

By HARRY STURGEON CROSSEN, M.D., F.A.C.S.

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# The American Journal of Obstetrics and Gynecology

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## Original Communications

### THE LIFE HISTORY OF OVARIAN HEMATOMAS (HEMORRHAGIC CYSTS) OF ENDOMETRIAL (MÜLLERIAN) TYPE*

*(From the Gynecological and Pathological Departments of the Albany Hospital and the Albany Medical College.)*

BY JOHN A. SAMPSON, M.D., ALBANY, N. Y.

NEXT to leiomyoma of the uterus, the pathologic conditions arising from the implantation of epithelium which escapes from the fallopian tubes into the peritoneal cavity, probably furnish the most frequent pelvic lesions found at operation in women between the ages of thirty and the menopause. I have encountered thirty-seven cases with these lesions during the year, May 1, 1921, to May 1, 1922, in 170 abdominal operations for pelvic disease in women between thirty and fifty years of age (in over twenty per cent of these patients); and six additional cases, three under thirty years of age and three over fifty. One of the latter had not reached the menopause. The epithelium primarily giving rise to these implantations is derived from or through the fimbriated ends of the fallopian tubes. It lodges either on the surface of the ovaries or on the peritoneal surface of the other pelvic structures, especially those in the culdesac, or on both the ovaries and the pelvic peritoneum, and develops into glands or tubules (adenomas) of endometrial (müllerian) type. The primary peritoneal implantation adenomas are usually small and insignificant, but may spread and become invasive. The implantations on the ovary invade the tissues of that organ, and as a result of their reaction

*Read in part at the Forty-seventh Annual Meeting of the American Gynecological Society, May 1-3, 1922.

NOTE: The Editor accepts no responsibility for the views and statements of authors as published in their "Original Communications."

to menstruation develop into superficial or deep hematomas (hemorrhagic or menstruating cysts) of endometrial (müllerian) type, which usually perforate into the peritoneal cavity. Perforation occurs in the superficial ovarian hematomas while they are still small, a few millimeters in diameter. On the other hand, the hematomas developing in the deeper tissues of the ovary may attain a much larger size, from 1 to 9 cm. in diameter before perforation occurs. The material escaping from the perforation of the ovarian hematoma, whether the latter is small or large, may carry with it epithelium which is cast off from its lining by menstruation. This epithelium may give rise to secondary implantations which are often apparently more invasive, and have a wider distribution than the primary (original) pelvic implants. The ovary may be looked upon as an intermediary host in the development of implantation adenoma of müllerian type but not as an essential one. The implantation adenomas of uterine and tubal (müllerian) type in the ovary may also, possibly, be a source of ovarian cysts and carcinoma.

All implantation adenomas of endometrial (müllerian) type should be of great pathologic interest, as they are sometimes of great clinical importance. This is especially true of the ovarian hematomas of endometrial type and the implantations arising from their perforation. It is the life history of these ovarian hematomas which I wish to present in this communication.

In a paper entitled Perforating Hemorrhagic (Chocolate) Cysts of the Ovary,[1] presented at the last meeting of the American Gynecological Society (June 3, 1921), I reported twenty-three cases of ovarian hematomas of endometrial type. All these cases occurred in my own practice, and fourteen of them were observed during the previous year. The hematomas described in this series varied in size from 1 to 9 cm. in diameter, most of them being from 2 to 4 cm. They were bilateral in eight of the twenty-three cases. A perforation had occurred in all of the twenty-three cases and was found on the lateral, or on the free surface of the ovary. In all specimens the tubes were apparently patent. At the operation the cyst or ovary was found to be adherent, and in freeing it the "chocolate" like contents escaped because a previous perforation, which had become sealed by whatever structure the ovary had become adherent to, was reopened or the wall of the cyst was torn. Adhesions were present in all cases and these varied greatly in location and extent. They were found about the ovary at the site of the perforation, in the natural pockets and folds of the pelvis where material escaping from such a perforation would be apt to lodge, and especially in the culdesac. When slight, they simulated the adhesions resulting from a pelvic peritonitis of tubal origin; but the adhesions in the culdesac

were sometimes accompanied by such a marked reaction as to resemble malignancy. A histologic study was made of the tissue involved in the adhesions in fourteen of the twenty-three specimens and aden-

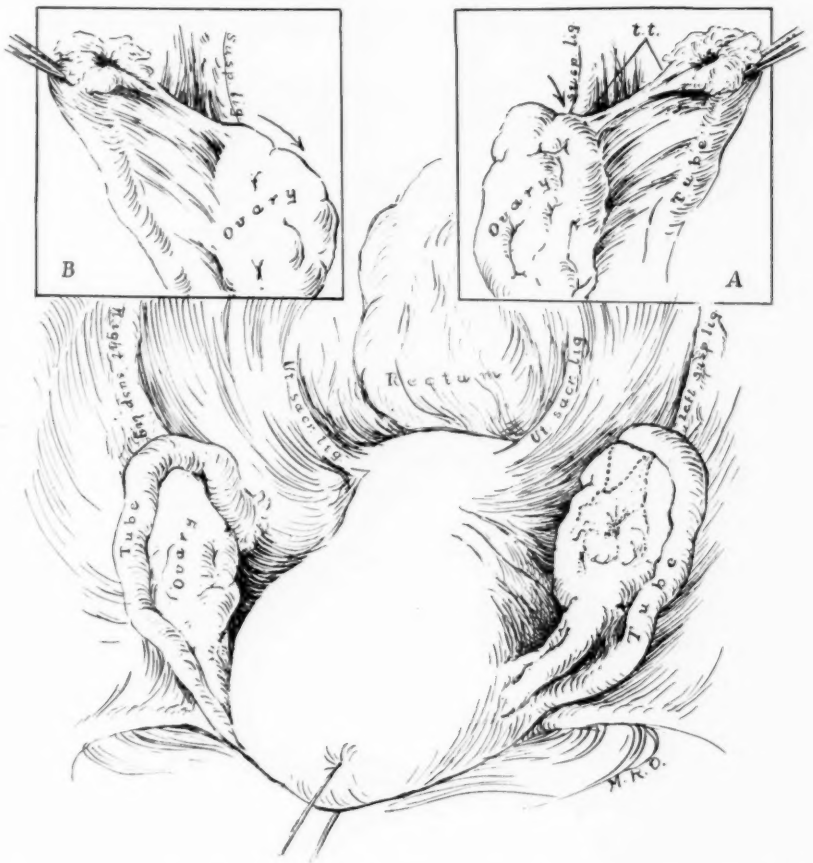


Fig. 1.—Two of the most frequent situations of the fimbriated ends of the normal fallopian tubes (x 5/6).

View of the pelvic contents as seen at operation: loops of the intestine lifted out of the pelvis, and the uterus drawn forward without disturbing the relation between the fimbriated ends of the tubes and the ovaries. The tube is usually longer than the distance from the cornu of the uterus to the distal pole of the ovary and, as the fimbriated end is freely movable, the latter is readily pushed into various situations by the contents of the pelvis. The distal pole of the ovary frequently projects beyond its attachment to the suspensory ligament in such a manner as to form a groove between it and the suspensory ligament (see arrow in insert A). The distal portion of the tube often slips into this groove and the fimbriated end of the tube is frequently situated beneath or lateral to the ovary, with its opening directed towards the surface of that organ by the "tether" like action of the free margin of the mesosalpinx (tubal tether, t.t.) to which the lower fimbriae of the tube are often attached. Should the distal pole of the ovary not project beyond the insertion of the suspensory ligament (see insert B) the fimbriated end of the tube slips down mesial to the ovary and is often tucked beneath that organ, the tubal "tether" (t.t.) directing the fimbriated opening towards the surface of the ovary. This mechanism of the "tubal tether" would facilitate the ovum's escape into the lumen of the tube in evulation: this same close anatomical relation between the fimbriated end of the tube and the surface of the ovary would also permit epithelium, escaping from the fimbriated end of the tube or through its lumen, to become implanted on the surface of the ovary. The various physiologic changes in the pelvic contents would also cause the fimbriated end of the tube to brush against the structures in the culdesac. Should implantation adenoma arise from epithelium escaping from the tube, we should expect to find these implantations most frequently on the lateral and under surface of the ovaries, and on the posterior surface of the lower portions of the broad ligament, the uterus and in the bottom of the culdesac; especially about the uterine attachments of the uterosacral ligaments. It is in these situations that the early implantation adenomas are most often found.

oma of endometrial type was found in thirteen of these. I concluded that these ovarian hematomas were a source of many of the adenomas of endometrial type, so-called adenomyomas, found in the pelvis and not continuous with the mucosa of the uterine cavity. The adenomas in these thirteen cases could have arisen from the implantation of epithelium carried with the contents of the ovarian hematoma escaping through the perforation. They were compared with the peritoneal implantations arising from the perforation of a malignant ovarian cyst. I added: "I cannot state that these ovarian hematomas of endometrial type are the only cause of ectopic pelvic adenomas."

I have discarded the term "perforating hemorrhagic cysts" as applied to this condition, because perforations may occur in other varieties of ovarian hematomas. I now refer to them as hematomas or hemorrhagic cysts of endometrial (müllerian) type. Their epithelial lining, where present, is similar to that found in the hematomas due to the retention of "menstrual" blood which occur in the

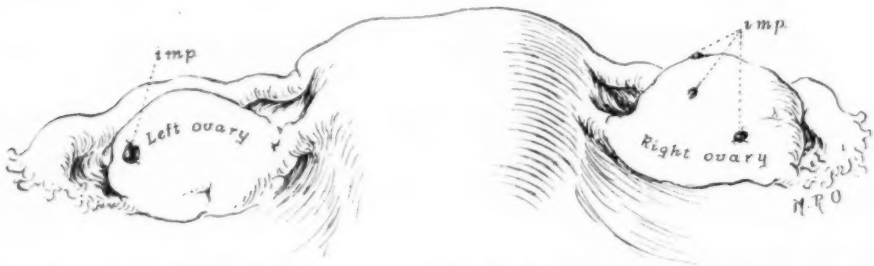


Fig. 2.—(Case 5). Implantation adenoma of endometrial type on the lateral surface of the left ovary, and the lateral and under surfaces of the right ovary. View of the posterior surface of the uterus and tubes, with the ovaries turned upwards, exposing their lateral surfaces (x 4/5). Implantation adenoma was found only on the surfaces of the ovaries as indicated. The lateral and under surfaces of the ovaries are the portions of the ovaries most frequently found in contact with the fimbriated extremities of the tubes (Fig. 1). The patient was menstruating the day of the operation, and the implantations appeared like miniature strawberries. For colored photomicrograph of a microscopic section of one of the adenomas shown on the surface of the right ovary, see Fig. 28. Fig. 29 shows a colored photomicrograph of a section through the adenoma on the surface of the left ovory.

adenomyomas of the uterus derived from the uterine mucosa, and the blood in the ovarian hematomas is also apparently of menstrual origin.

In the first communication I described small hemorrhagic areas in the ovaries of three patients who had been operated upon during their menstrual period. Histologically these areas proved to be due to hemorrhage about or into a space lined by tissue of endometrial (müllerian) type. I stated that I believed these gland-like spaces were lined by epithelium of endometrial type as shown by their structure and by their function (menstruation). I also stated that the ovarian hematomas described in that paper might have arisen from these endometrial-like structures. At the time the paper was sent to the Archives of Surgery for publication I had not been able completely to

trace the development of the ovarian hematomas of endometrial type from these structures, but during the month of August, 1921, I did so. A note was added to my paper as it appears in the Transactions of the American Gynecological Society stating that all the conditions found in the hematomas described in that paper had been traced from these endometrial-like glands which reacted to menstruation. It was also suggested in this note that the gland-like structures in the ovary, from which these hematomas develop, might arise from the implantation of epithelium derived from or escaping through the fallopian

Fig. 3.—(Case 18). Implantation adenoma of endometrial type on the posterior surface of the lower portion of the right broad ligament, (*a*), the culdesac lateral to the rectum (*b*) and the posterior surface of the uterus (*c*). View of the pelvis from above with the uterus drawn upward (from sketch made at the operation, x 5/6). The uterus was retroflexed, and on drawing it forward these implantations were observed. On replacing the uterus in retroflexion it was noted that the adenoma (*c*) came in contact with adenoma (*b*) and I believe that it was probably a contact implantation from adenoma (*b*). I believe adenomas (*a* and *b*) arose from epithelium escaping from the right fallopian tube. For the gross appearance of the adenomas (in colors) see Figs. 17, 18, and 19, and for the histologic structure of adenoma (*b*) see Fig. 24. The patient was operated upon the day after menstruation had ceased.

tubes and that implantation adenoma in the pelvis might arise from this source as well as from the perforation of an ovarian hematoma.

At a meeting of the Interurban Surgical Society at Albany, N. Y., November 25, 1921, I presented the pathologic findings in twelve cases[2] in which portions of the intestinal tract were involved by implantation adenomas of endometrial type which could have arisen from epithelium escaping from a perforated ovarian hematoma. The

development of the hematomas from the gland-like structures of endometrial type in the ovary was also demonstrated at this meeting.

On February 14, 1922, before the Harvard Medical Society at Boston, Mass., I reviewed in a general way the entire subject of ovarian hematomas and implantation adenomas of endometrial type.[3] As stated at that meeting my interpretation of the origin and development of these implantation adenomas was as follows: Tubal and uterine epithelium at times escapes into the peritoneal cavity from or through the fimbriated end of the tube. It lodges where such material would be likely to fall, especially on the lateral surface of the

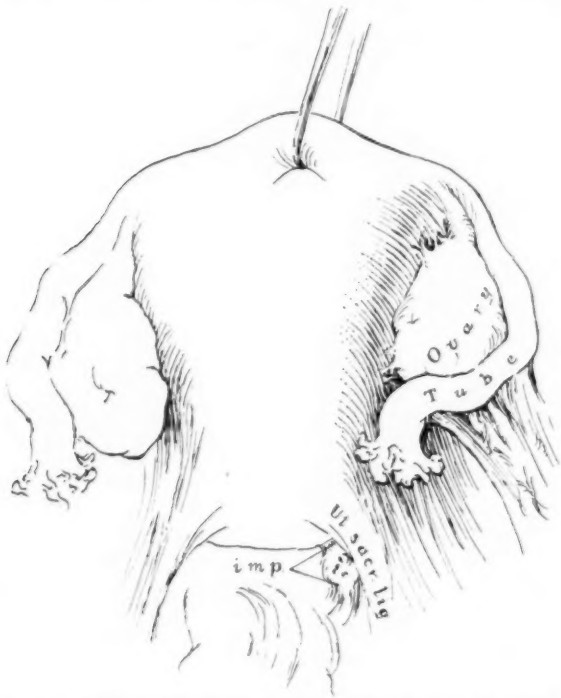


Fig. 4.—(Case 13). Implantation adenoma of endometrial type in the culdesac, mesial to the uterine attachment of the right utero-sacral ligament. View of the uterus, tubes and ovaries from above, uterus drawn upward and forward (from sketch made just after the operation, x 5/6). The uterus was retroflexed, and on drawing it forward a small hard nodule with pigmented dots on its surface was detected in the culdesac, as indicated. This was removed (see Fig 13). The right ovary was lightly adherent to the posterior surface of the uterus, broad ligament and the side of the pelvis. The ovary was freed and I was unable to detect any evidence of adenomas on its surface. The tubes were patent. I believe the adenoma in the culdesac could have arisen from epithelium escaping from the tube, although it might have arisen from a small hematoma of the ovary which had disappeared after perforation.

ovary, on its free border and in the culdesac, the distribution corresponding to the distribution of pus escaping from the fallopian tubes in salpingitis. Adenoma may develop wherever this epithelium falls on suitable "soil." We may, therefore, have implantations only on the ovary, especially on its lateral surface and free border, or both on the ovary and in the pelvis or in the pelvis alone. The tubules arising in the ovary may develop into hematomas which usu-

ally perforate and give rise to implantation adenomas, often apparently more invasive (virulent) and with a wider distribution than the implantations found without evidence of an ovarian hematoma. I consider the ovary as a sort of intermediary host, hot bed or incubator which sometimes imparts greater virulence to the epithelial cells developing in it, but it may not be an essential intermediary host in the origin of implantation adenomas of endometrial type. In this same communication the development of ovarian cysts and carcinoma from these tubules of endometrial type was also discussed.

I have used the term ovarian hematomas of endometrial type rather than endometrial hematomas because I believe that in some instances the epithelium lining them may possibly be derived primarily from the tubal mucosa. The term müllerian would be inclusive and a bet-

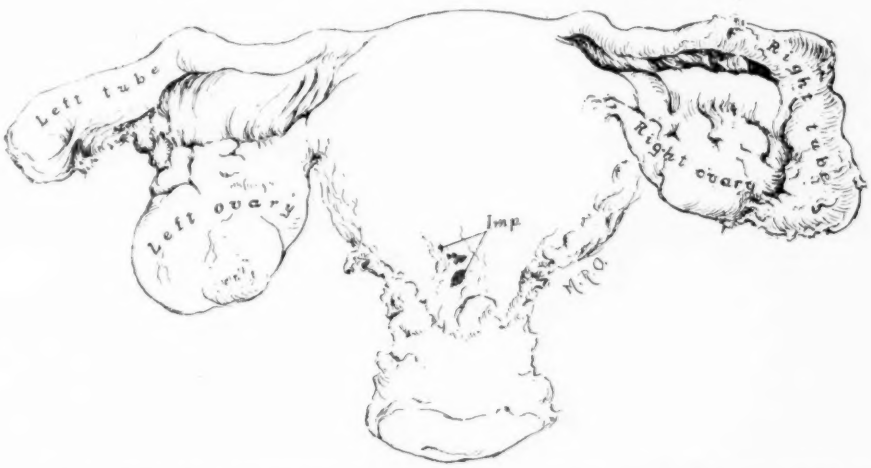


Fig. 5.—(Case 12). Implantation adenoma of endometrial type, of the posterior surface of the uterus (see also Figs. 12 and 20); bilateral hematosalpinx, adenoma formation in the fimbriated ends of both tubes, with extension of the tubal mucosa to the peritoneal surface of the distal ends of the tubes; leiomyoma of the uterus. Posterior view of the uterus, tubes and ovaries (x 2/3). Hemorrhage was found in the stroma of the walls of the dilated tubules of the adenoma of the tubes, and this indicated that the hematosalpinx arose from this source. The adenoma invading the posterior surface of the uterus was similar, in its histologic structure, to the glandular structures of the adenoma in the distal portion of the tube. (Figs. 11 and 12.) The situation of the adenoma of the posterior surface of the uterus and its structure indicate that it could have arisen from the implantation of epithelium escaping from the tube. Adenoma of endometrial type was not found in the ovaries.

ter one. Is it possible to decide whether a given implantation adenoma was primarily derived from tubal or uterine mucosa? I do not think it can be definitely proved in any instance but the histologic structure of some of these adenomas suggests a tubal, and others a uterine, origin.

Adenomyoma of the uterus and of the tube may be classified as either primary or secondary. In primary adenomyoma the adenomatous growth is derived from the direct invasion of the uterine or tubal wall by the mucosa lining their cavities. In the secondary type of growth, which in my experience is the much more frequent vari-

ety, the uterine or tubal wall is invaded by epithelium implanted on its peritoneal surface, which is derived from the perforation of an ovarian hematoma of endometrial type or from or through the fimbriated extremity of the tube. It is obviously possible that the epithelium giving rise to a secondary adenomyoma of the uterus might be derived primarily either from the tubal or the uterine mucosa, and the same is true of secondary adenomyoma of the tube.

A comparative microscopic study of primary adenomyoma of the tube and of the uterus demonstrates that there is frequently a very evident distinction between the two. The adenomatous growth derived from the tubal mucosa consists of tubules and dilated tubules lined by epithelium, usually without the characteristic stroma and glands found in normal endometrium; or if the latter are present they are insignificant as compared with the general histologic picture of

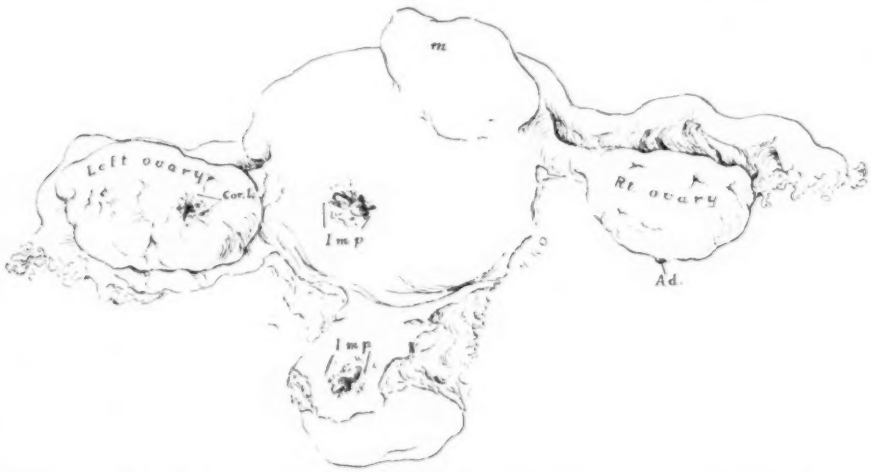


Fig. 6.—(Case 17). Implantation adenoma of endometrial type of the posterior surface of the uterus, the culdesac mesial to the left utero-sacral ligament, and the under surface of the right ovary; corpus luteum hematoma of the left ovary, multiple leiomyomas of the uterus. Posterior view of the uterus, tubes and ovaries (x 2/3). The uterus was retroflexed, and the adenoma on the posterior surface of the uterus was in contact with the one in the culdesac, and probably is a contact implantation from the latter (Fig. 14). It is possible that the adenoma involving the surface of the ovary (Fig. 15) may represent the remains of a small hemorrhagic cyst, which had perforated and lost the greater portion of its epithelial lining; and that the other implantations arose from epithelium escaping from this source. It is more likely that the ovarian and peritoneal implantations had a common origin from epithelium escaping from or through the tubes, which were patent.

the growth. In primary adenomyoma derived from the uterine mucosa the histologic picture is reversed, the prevailing type of growth resembles, with its stroma and gland formation, normal endometrium; while the tubules and dilated tubules so characteristic of a tubal adenoma are lacking or only form a small part of the entire growth. If only isolated areas are examined microscopically it may be impossible to distinguish between the two, but if the growth is studied as a whole the distinction is usually evident. A comparative study of the epithelium lining the uterus and the tube, apart from the under-

lying stroma, is of interest. The height of the epithelium lining a uterine gland usually varies with the dilatation of the gland; the greater the dilatation of the gland the lower the epithelium. The height of the uterine epithelium is usually more uniform than that of tubal epithelium, except in glandular hypertrophy, and even then there may not be as great a tendency for the formation of "tufts," which are due to a localized heaping up of epithelium with a subsequent increase in height as is so often seen in the ampulla of the tube. Cilia are usually more readily demonstrable on tubal than on uterine epithelium. Primary adenomyoma of the tube generally arises in the isthmus of the tube, where the epithelium more closely resembles that

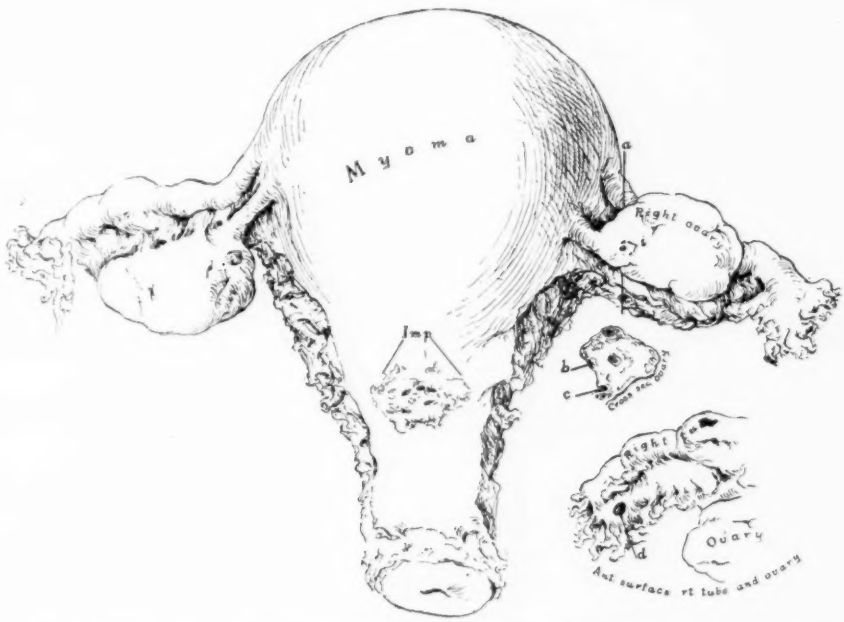


Fig. 7.—(Case 16). Implantation adenoma of endometrial type of the posterior surface of the uterus, lateral surface of the right ovary, and of the right fallopian tube near its fimbriated extremity; leiomyoma of the uterus. Posterior view of the uterus, tubes and ovaries (x 1/2). The adenoma (imp.) on the posterior surface of the uterus was situated at about the junction of the cervix and body of the uterus. The right ovary is turned upwards with its lateral surface exposed, showing the situation of the adenomas at (*i*). The adenomas on the surface of the ovary are also indicated by (*b*) and (*c*) of the cross section of the ovary, through the plane (*a*). The situation of the adenoma on the anterior or lateral surface of the right tube is indicated by (*d*) of the insert. Histologically the adenomas on the surface of the uterus, ovary (Fig. 16) and tube were similar, and I believe could have had a common origin from epithelium escaping from the right tube, as their distribution suggests.

of the endometrium than does the epithelium situated in the ampulla, but even in these adenomyomas we frequently find that the dilated tubules attempt to reproduce the structure of the mucosa of the tube, so that sometimes a careful microscopic study of the stained section may be required to distinguish the lumen of the tube from some of the dilated tubules in its wall. In the study of ectopic adenoma of possible endometrial or tubal type we must bear in mind normal endo-

metrium, glandular dilatations and hypertrophies, primary adenomyoma of the uterus, and especially the hematomas arising in these. We must likewise consider the normal tubal mucosa; that found in hydro- and hemato-salpinx and the structures in primary adenomyoma of the tube.

The first communication was based on the study of twenty-three cases of ovarian hematomas (hemorrhagic cysts) of endometrial type,

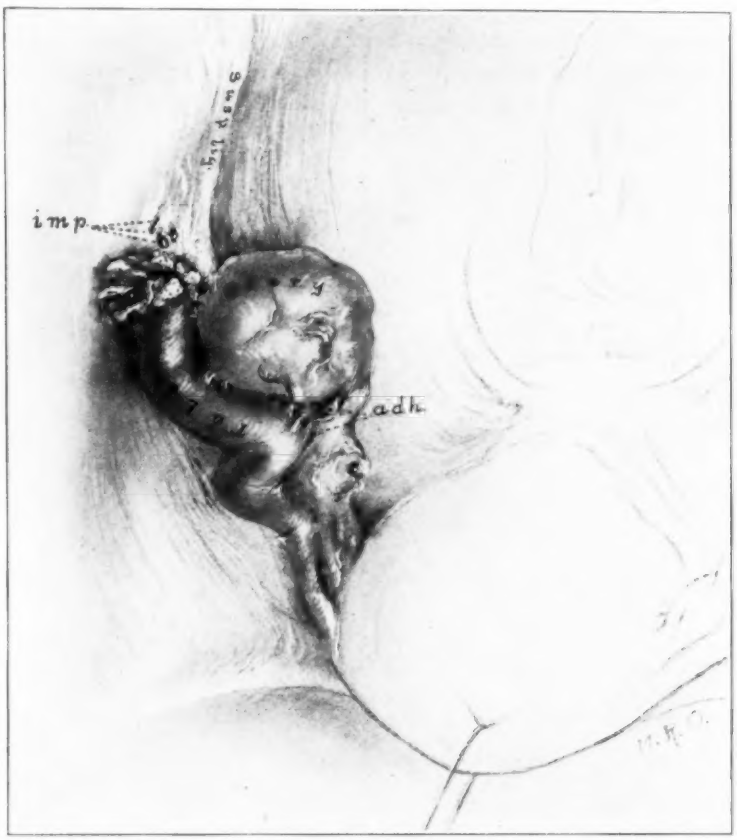


Fig. 8.—(Case 19). Implantation adenoma of endometrial type of the anterior surface of the suspensory ligament of the right ovary, and also of the mesial surface of the ovary near its attachment to the broad ligament; adhesions between the tube and ovary, uterus retroflexed. The condition found at operation is shown after replacing the uterus and drawing it forward, without disturbing the tube and ovary (from sketch made at the operation, x 1). As indicated, the tube was kinked, and adherent to the upper surface of the ovary in such a manner that the fimbriated extremity rested on the anterior surface of the suspensory ligament. An adenoma, histologically similar to the one shown in Fig. 16, was situated on the anterior surface of the suspensory ligament directly in front of the opening of the tube. Should epithelium escape from the tube, while in this situation, we should expect implantation adenoma to develop on the mesial rather than on the lateral surface of the ovary. Adenoma was found on the mesial, and not on the lateral surface of the ovary.

eight of which were bilateral. From May 1, 1921, to May 1, 1922, I have collected thirty-three additional cases of ovarian hematomas, all but three occurring in my own practice. Four of the thirty-three

specimens were removed prior to May 1, 1921, and had been previously preserved as doubtful specimens.

I have collected and studied fifteen cases of implantation adenoma in the pelvis in which the implantations were apparently of tubal (from or through) rather than of ovarian origin as will be discussed later.

The clinical histories of twenty cases are reported in the present paper and these were chosen from cases demonstrating the origin and development of the ovarian hematomas rather than from the more advanced ones with perforation and extensive implantations.

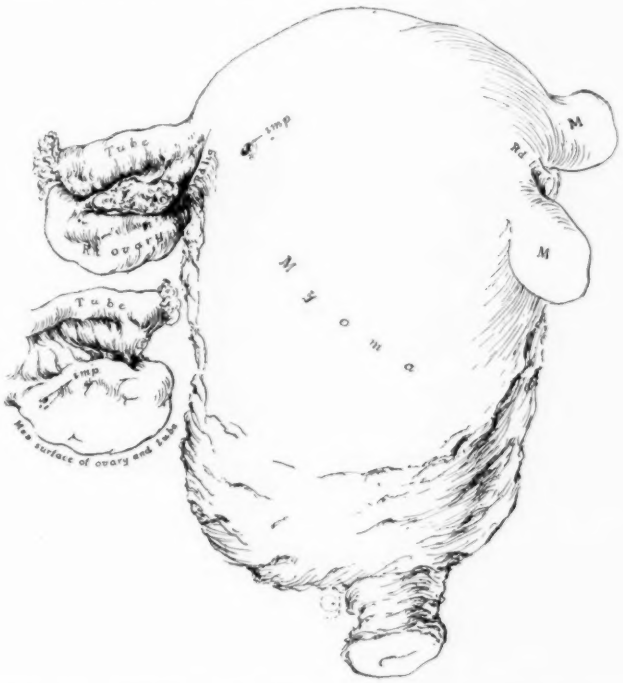


Fig. 9.—(Case 15). Implantation adenoma of endometrial type of the anterior surface of the uterus mesial to the attachment of the right round ligament and also on the mesial surface of the right ovary; multiple leiomyomas of the uterus. Anterior surface of the uterus, right tube and ovary (x 1/2). The situation of the adenoma on the anterior surface of the uterus is indicated (imp.) and also the one on the mesial surface of the right ovary (see imp. of the insert). The histologic structure of the latter was similar to that shown in Fig. 16. The tube was short and the pelvis was filled by the enlarged uterus, and therefore epithelium, escaping from the tube, would be more apt to lodge on the mesial surface of the ovary than on the lateral. If the epithelial implantations were the result of a retrograde menstruation, or there was any free fluid in the peritoneal cavity, some of the epithelium might become lodged on structures at a distance from the tube.

## THE ORIGIN OF THE TUBULES OF ENDOMETRIAL (MÜLLERIAN) TYPE IN THE OVARY FROM WHICH HEMATOMAS OF ENDOMETRIAL (MÜLLERIAN) TYPE ARISE

The most natural conception of the source of these tubules is from an abnormal development of the surface epithelium of the ovary or from developmentally misplaced epithelium of the müllerian duct.

Theories to this effect have been presented by Russell,[4] Casler,[5] Norris[6] and more recently by Janney.[7] It is difficult to disprove any of these theories. The implantation theory which I have presented,[2] that these tubules arise from epithelium (possibly both tubal and uterine) escaping from or through the tube, is based on the following data:

Epithelium is found on the surface of the ovaries in these specimens, invading the underlying tissue as tubules. The epithelium lining these tubules is often ciliated; sometimes the tubules suggest a tubal origin and at other times a uterine. The ovarian tissue about the epithelium on the surface of the ovary as well as that about the tubules in the deeper portions, sometimes reacts to menstruation.

In some cases minute adenomas of apparently the same age are found only on the surface of the ovary, in others both on the surface

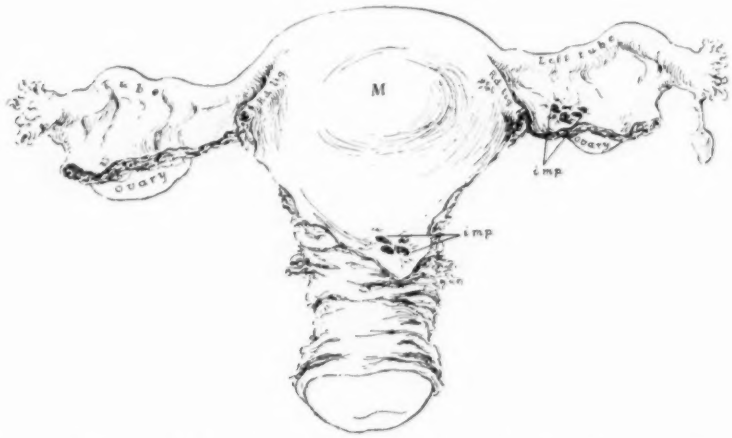


Fig. 10.—(Case 4). Implantation adenoma of endometrial type of the posterior surface of the uterus near the attachment of the left broad ligament, the anterior surface of the left broad ligament and the vesico-uterine fold of peritoneum; leiomyomas of the uterus. Anterior view of the uterus (x 1/2). The implantation adenomas of the anterior surface of the left broad ligament and the vesico-uterine fold are indicated (imp.). A pigmented elevation about 4 mm. in diameter was found on the lateral surface of the right ovary. Histologically it consisted of pigmented cells (old hemorrhage) without any epithelium. It might represent the remains of a hematoma of endometrial type, in which all of the epithelial lining had been cast off by menstruation, and the implantation adenomas might have arisen from this source. The epithelium causing the implantations might have escaped from the tubes, having been carried to distant portions of the pelvis by such factors as a profuse retrograde menstruation, or free fluid in the peritoneal cavity. Usually the implantations, apparently arising from or through the tubes, are situated in places which are or might have been in contact with the fimbriated ends of the tubes as shown in previous illustrations. Even a small amount of fluid escaping from the tubes, as well shown in some cases of purulent salpingitis and tubal pregnancy, may reach other portions of the pelvis than that about the fimbriated opening of the tube.

of the ovary and on the pelvic peritoneum, and in still others only on the latter. These are all most frequently present in places where material escaping from the tube would be most apt to lodge (Figs. 2 to 10 inclusive), and suggest a common origin from the latter.

These hematomas are unusual in women under thirty years of age and if they were of developmental and not of acquired origin we would expect them to occur in younger women, soon after puberty.

They develop during the menstrual life of the patient, when tubal

and uterine epithelium would be more likely to escape from or through the fimbriated end of the tube than before puberty and after the menopause. It is possible that the implantation on the ovary of epithelium derived from or through the tube may occur before puberty and after the menopause, and may develop into ovarian cysts or even carcinoma.

The uterus in these cases is often retroflexed, contains leiomyomas and polyps, conditions which might favor a back flow of menstrual blood through the tube.

In the fifty-six cases of ovarian hematoma of this type which I

Fig. 11.—(Case 12). Cross section (x 8) of the distal end of the left fallopian tube shown in Fig. 5. Bilateral hematosalpinx, with adenomatous structure of the distal end of both tubes was present, possibly originating from a folding in of the fimbriae of the tubes. The glands and dilated glands or tubules in this section resemble, histologically, the glands and tubules found in some of the implantation adenomas of endometrial type. Hemorrhage was present in places beneath the epithelial lining of some of the tubules; in other places the epithelial lining was absent, apparently having been cast off by the hemorrhage rupturing into the cavity of the tube. Endothelial leucocytes were also present. The hemorrhagic contents of the hematosalpinx evidently arose from this source; a probable reaction to menstruation. Some of the hemorrhagic contents of the hematosalpinx including epithelium cast off by menstruation might have escaped, at one time, into the peritoneal cavity, and could have been the source of the implantation adenoma on the posterior wall of the uterus; shown in Figs. 5 and 12.

have studied, the tubes were apparently patent in all, suggesting that this source of implantation was open in all of these cases. The presence of occluded tubes would not exclude the origin of the ovarian implantations from this source as they might have occurred before the tubes had become closed. In the fifteen cases of pelvic implantations apparently not derived from a perforated ovarian hematoma the

tubes were patent in all but one instance. In that case (case 12 of this series) bilateral hematosalpinx was present with "adenomyoma" of the fimbriated ends of both tubes and the extension of the adenoma to the peritoneal surface of the tubes (Figs. 5, 11 and 12). The implantation adenoma of the posterior surface of the uterus resembled histologically the adenoma in the tube, and was found at the operation in close proximity to the end of one of the tubes, from which I believe it arose.

Implantations of endometrial type on the peritoneal surface of the pelvic structures apparently arise from the escape of the contents of an ovarian hematoma of this type and furthermore similar implantations occur on the surface of the ovary about the perforation,

Fig. 12.—(Case 12). Photomicrograph (obj. 16 mm.) of a section of a portion of the implantation adenoma of the posterior surface of the uterus shown in Fig. 5. Histologically it resembles the glands and dilated glands or tubules shown in Fig. 11. I believe that it could have arisen from the implantation of epithelium escaping from the fallopian tube as stated in the legend of Fig. 11.

and possibly also on the opposite ovary. The implantations involving the ovaries from this source simulated the original ovarian implantations, but were apparently often more virulent, and more closely resembled typical endometrium in those specimens studied. As implantations arise from the perforation of the ovarian hematomas so may ovarian hematomas arise from implantations. The most obvious source of the latter in the absence of an ovarian hematoma with evidence of perforation is from or through the fallopian tubes.

These ovarian hematomas are often bilateral, and usually develop and perforate on the lateral or the under surface of the ovaries, the portions of the ovaries which are anatomically most frequently in contact with the fimbriae of the tubes (Fig. 1), as will be discussed later.

The escape of the ovum from the ovary into the fallopian tube during ovulation is an evidence of the close contact of the opening of the tube and its fimbriae with the surface of the ovary. This same close contact would favor the implantation of epithelium on the surface of the ovary from the fimbriae of the tube or from epithelium escaping through its lumen.

We have abundant proof that apparently normal uterine and tubal epithelium may be invasive, as demonstrated in the development of "adenomyoma" from the invasion of the uterine and tubal wall by the epithelium lining their cavities.

As has been already stated, implantation adenoma may also arise from the perforation of ovarian hematomas of endometrial type, as

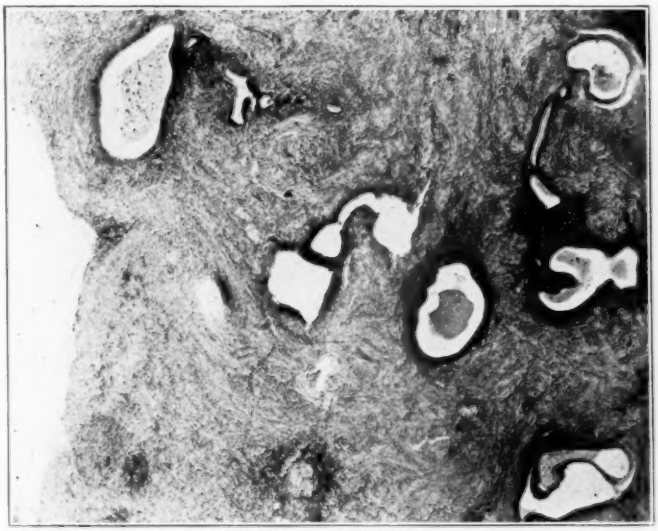

Fig. 13.—(Case 13). Photomicrograph (obj. 50 mm.) of a section of the adenomatous nodule situated mesial to the uterine attachment of the right utero-sacral ligament shown in Fig. 4. It is an adenoma of endometrial (müllerian) type resembling the adenoma shown in Fig. 12. It could have arisen from epithelium escaping from or through the fallopian tube as indicated in Fig. 4. It is impossible to state whether this epithelium was derived from the tubal or the uterine mucosa. Histologically it could have arisen from either, but possibly it suggests more closely a tubal origin.

shown in my first communication (possibly not yet accepted by others).

Why is not every cesarean section, hysterotomy, salpingectomy and ruptured tubal pregnancy followed by implantation adenoma? Conditions may not always be suitable for the implantation of epithelium. Dr. F. B. Mallory of Boston, Mass., in a personal communication to me, states that he has examined two specimens of adenoma of endometrial type found in the scar of the abdominal incision after cesarean section.

During the last year, Dr. Victor C. Jacobson has experimentally proved, in the pathological laboratory of the Albany Hospital and

the Albany Medical College, that the uterine mucosa of the rabbit may be transplanted and give rise to adenoma of endometrial type resembling those described by me except for the absence of any reaction to menstruation; furthermore some of the transplantations made by him in the ovary developed into "ovarian cysts." The results of these experiments have been reported by him under the title "The Autotransplantation of Endometrial Tissue in the Rabbit."[8]

Since September, 1921, I have been studying the situation of the fimbriated end of the normal fallopian tube as found at operation. With the patient in the Trendelenburg position the uterus, tubes and ovaries were carefully exposed without disturbing their relation to each other; and the exact situation of the fimbriated ends of the tubes was observed. In several instances sketches demonstrating the con-

Fig. 14.—(Case 17). Photomicrograph (obj. 50 mm.) of a section of the implantation adenoma in the culdesac shown in Fig. 6. Histologically it resembles uterine mucosa, and suggests either that the epithelium giving rise to it resulted from a retrograde menstruation through the tube, carrying some of the uterine epithelium with it; or if it arose from tubal epithelium that the latter developed into a structure simulating uterine mucosa.

ditions found were made at the operation by Miss Oliver. The position of the fimbriated end of the normal tube depends upon many factors, such as the length of the tube, the length of its mesentery at the fimbriated extremity, and the condition of the surrounding parts. As the fimbriated end of the normal tube is freely movable, it will naturally be pushed or slipped into places where it will be the least in the way. The length of the tube is usually greater than the distance from the cornu of the uterus to the distal pole of the ovary, so that its fimbriated end is usually found behind the ovary or below and mesial to it. The distal pole of the ovary frequently extends beyond the attachment of its suspensory ligament in such a way as to form a groove between it and the suspensory ligament. The distal portion of the fallopian tube easily slips into this groove, and the fimbriated end

of the tube lies between the ovary and the side of the pelvis, or beneath the ovary, often with the opening directed towards the ovary, due to the tether-like action of the distal portion of the mesosalpinx (Fig. 1). If this groove is shallow or absent the distal portion of the tube usually hangs down in the pelvis (over) mesial to the ovary in such a way that the tubal fimbriae are in contact with the structures in the bottom of the culdesac (Fig. 1). Even in this latter group the fimbriated end of the tube is often found tucked beneath the ovary. These are the two most frequent situations of the tube but occasionally it is found in others (Figs. 8 and 9). Material (including epithelium) escaping from the fimbriae of the tube or through its lumen, would be apt to lodge on the structures in contact with the fimbriae of the tube. If implantations of epithelium should arise from this source we would expect to find them on the lateral and the under surface of the ovary and in the culdesac, especially about the utero-

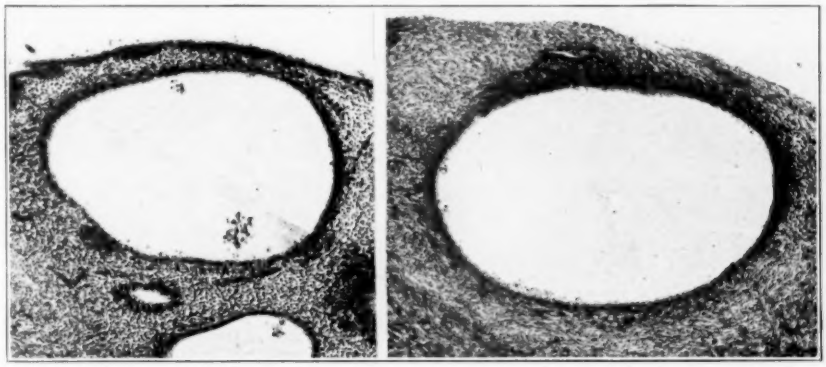

Fig. 15.—(Case 17). Photomicrograph (obj. 16 mm.) of a section of the adenoma of the right ovary indicated in Fig. 6 and also of uterine mucosa with glandular dilatation for comparison. The photomicrograph of the uterine mucosa is shown to the left and that of the adenoma of the ovary to the right. A dilated "gland" with a small gland (*g*) above it appears in this section of the adenoma of the ovary. Hemorrhage is present in the ovarian tissue about the dilated gland, giving rise to the "blueberry" appearance of the surface of the ovary over the adenoma as shown in Fig. 21. The epithelial lining of the dilated gland of the ovarian adenoma was identical with that of the dilated gland in the uterine mucosa; low to cuboidal, the latter predominating.

sacral ligaments and on the posterior surface of the broad ligament. It is in these situations that the early (primary) implantation adenomas are most frequently found. The implantation adenomas seem to thrive in ovarian tissue and smooth muscle. As women spend the greater portion of the twenty-four hours of the day with the body in the upright posture, whether sitting down or standing, the tendency for the fimbriated end of the tube to be tucked beneath, or lateral to, the ovary would be increased, and "sediment" escaping from the tube would naturally settle on the lateral and the under surface of the ovary and in the bottom of the culdesac, especially its anterior portion.

Implantation adenoma may occur on the ovary alone, especially on

its lateral or its under surface, or on the surface of the ovary and in the pelvis, especially the culdesac, or in the pelvis alone. The pelvic implantations are usually small but may spread and become invasive. The ovarian implantations usually develop into hematomas of endometrial type which sometimes attain a large size, 6 to 9 cm. in diameter. Whether the ovarian hematomas are large or small, perforation usually occurs and implantation adenomas arise in the pelvis, which, in their distribution, correspond to the implantations of carcinoma from the perforation of a malignant ovarian cyst. They may occur

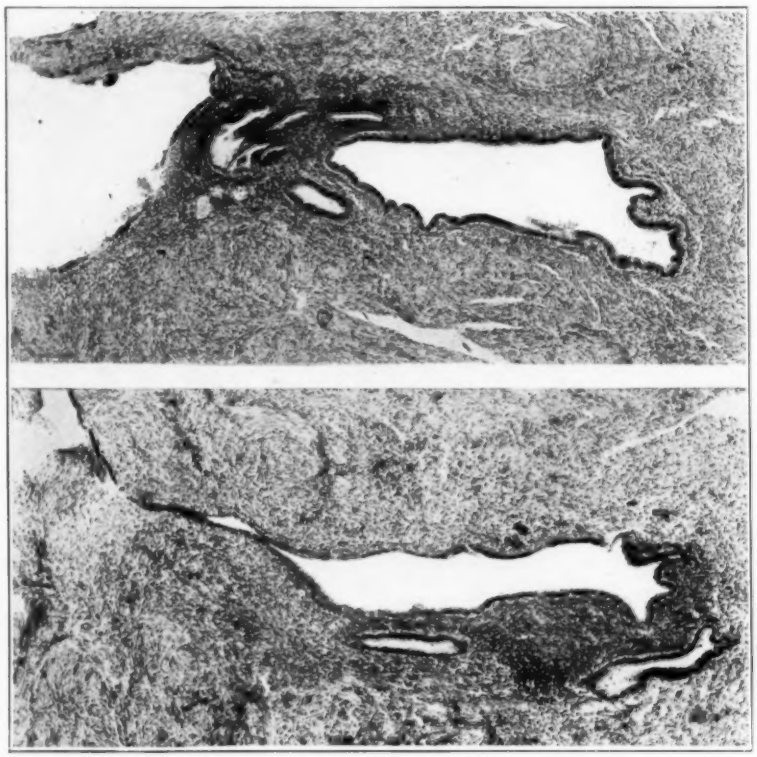

Fig. 16.—(Case 16). Photomicrographs (obj. 16 mm.) of the adenomas of endometrial (müllerian) type invading the lateral surface of the right ovary and the posterior surface of the uterus, as indicated in Fig. 7. The lower one is ovarian and the upper one uterine. Their histological structure is similar, and I believe that they both have a common origin from epithelium escaping from or through the fallopian tube. It is impossible to decide, from their structure, whether the epithelium was derived from the tubal or the uterine mucosa.

on any of the pelvic structures, including the surface of the ovary about the site of the perforation.

The relation between ovarian hematomas of endometrial type and implantation adenomas of the same type in the pelvis is very interesting. I have had three cases of typical ovarian hematoma without gross evidence of perforation, and was unable to find any evidence of adhesions or implantations in the pelvis. I believe that the subsequent perforation of the hematomas in these three cases would prob-

ably be followed by implantations in the pelvis. In forty-three cases of ovarian hematomas with evidence of perforation, in which the adhesions apparently resulting from the escape of the contents of the hematoma were studied microscopically, implantation adenoma of endometrial type was found in some portion of this tissue in all but one instance. The larger the hematoma, and apparently the greater the size of the perforation, usually the greater the distribution of the implantations. The pelvic implantations could have arisen from two possible sources in these cases: first, as primary implantations from or through the tubes, the source of the adenomas of the ovary or ovaries from which the ovarian hematomas arose, and secondly, as secondary implantations from the perforation of the ovarian hematoma. In some of these cases implantations which probably arose from both sources were present. In other cases the implantations may have arisen only from one source, either the tube or the ovary. I believe that when there is evidence of an ovarian hematoma with perforation, the hematoma is usually the principal and sometimes the only source of the accompanying implantations. All specimens of extensive implantation adenomas which I have studied have been associated with an ovarian hematoma of endometrial type, with evidence of perforation as in Figs. 61 and 62.

I have studied fifteen cases of implantation adenoma in the pelvis in which the implantations were apparently of tubal (from or through) rather than of ovarian origin, although the latter source cannot be positively excluded, as small ovarian hematomas may sometimes disappear after perforation. The implantations in all these cases were small as in Figs. 3, 4 and 5. Seven of these fifteen cases are reported in full in this communication and eight of them were mentioned in a previous paper.[3] One or both ovaries were removed in all but two cases. In the ovaries of nine of the thirteen cases which were examined, tubules of müllerian type were found, which could be interpreted as the possible remains of a small ovarian hematoma that had perforated, but I am more inclined to consider them as tubules which had not yet developed into hematomas. It was interesting to note the character of the implantations when there was no gross evidence of an ovarian hematoma with perforation. They were usually smaller, less invasive and not as widely distributed as those generally found in the pelvis associated with an ovarian hematoma with evidence of perforation. (Compare Figs. 3, 4 and 5 with Figs. 61 and 62.) Many of them also presented a little different histologic picture; they usually did not resemble typical endometrium as closely as did the implantations which were associated with an ovarian hematoma with perforation. The study of these fifteen cases suggested that the epithelium from which the implantations arose was derived from the tubal mu-

cosa in some, and from the uterine mucosa (menstruation with a back flow through the tubes) in others. In the latter group they may have arisen from a portion of tubal mucosa which had reacted to menstruation. The implantations in nearly all cases apparently reacted to menstruation, which might be interpreted as evidence against the epithelium being derived from the mucosa of the tube; but this function may have been acquired after implantation. The tubules in the ovaries from which the hematomas arise at times suggest a tubal origin, and at other times a uterine origin; but the implantations which apparently arise from the perforation of the ovarian hematoma, while they sometimes resembled the glandular elements of both tubal and uterine adenomyoma in the same case, more often resembled those of uterine adenomyoma. These observations suggest that the ovarian implantations from which the hematomas arose may have been derived both from tubal and uterine epithelium; but even if of tubal origin, by its growth in the ovarian hematoma it was converted into structures of uterine type.

In all, sixty-five specimens containing ovarian hematomas (hemorrhagic cysts) in various stages of development (this includes nine specimens with adenomas which had not yet developed into hemorrhagic cysts) and retrogression have been studied, eighteen of these being bilateral. In many instances multiple hematomas were present in the same ovary. Many were small, ranging in size from those just visible to the naked eye to those a few millimeters in diameter. Most of the larger ones varied in size from 1 to 4 cm. in diameter

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PLATE I

Fig. 17.—Plate I (Case 18). The appearance of the surface of the implantation adenoma in the culdesac (*b*) Fig. 3, (natural size). The "red raspberry" appearance is due to recent hemorrhage, the last menstruation having ceased the day before the operation. For histologic structure of the implantation see Fig. 24.

Fig. 18.—Plate I (Case 18). The appearance of the implantation adenoma on the posterior surface of the right cornu of the uterus, (*c*) Fig. 3, (natural size). It is possibly a contact implantation from the adenoma in the culdesac, (uterus retroflexed) as explained in the legend of Fig. 3. Like the preceding, it has a strawberry or red raspberry appearance; its histologic structure is also similar.

Fig. 19.—Plate I (Case 18). The appearance of the implantation adenoma on the posterior surface of the right broad ligament, (*a*) Fig. 3, (natural size). It resembles a blueberry in coloring. Apparently there was very little reaction to the last menstrual period; old hemorrhage was present giving it a darker coloration than the preceding.

Fig. 20.—Plate I (Case 12). The appearance of the implantation adenoma on the posterior surface of the uterus (Figs. 5 and 12) (natural size). The last menstruation occurred three weeks before the operation. The blood contained in the adenoma was old, hence the "blueberry" appearance of the elevations on the surface.

Fig. 21.—Plate I (Case 17). The appearance of the adenoma on the under surface of the right ovary (natural size). See also Figs. 6 and 15. The last menstruation occurred a week before the operation. The surface of the adenoma has the "blueberry" coloration due to old hemorrhage in the ovarian tissue.

Fig. 22.—Plate I (Case 20). The lateral surface of the left ovary, showing implantation adenoma: *"ad"* which appears red due to recent hemorrhage (natural size). The patient was due to menstruate the day of the operation. For histologic structure of the adenoma see Fig. 23.

Fig. 23.—Plate I (Case 20). Reproduction of a colored photomicrograph of a section of the adenoma shown in Fig. 22, H. & E. stain (obj. 16 mm.). A dilated gland or tubule lined by columnar epithelium is present. This epithelium is surrounded by a recent hemorrhage (premenstrual) in the ovarian tissue about it. Hemorrhagic cysts or hematomas of endometrial type arise from these adenomas (Figs. 25 to 29). The adenomas arise from the implantation of epithelium escaping from the tube or from other implantations; especially from the perforation of an ovarian hematoma of endometrial type. In this case I believe the epithelium came from or through the tube.

Fig. 17

Fig. 18

Fig. 19

Fig. 20

Fig. 21

Fig. 22

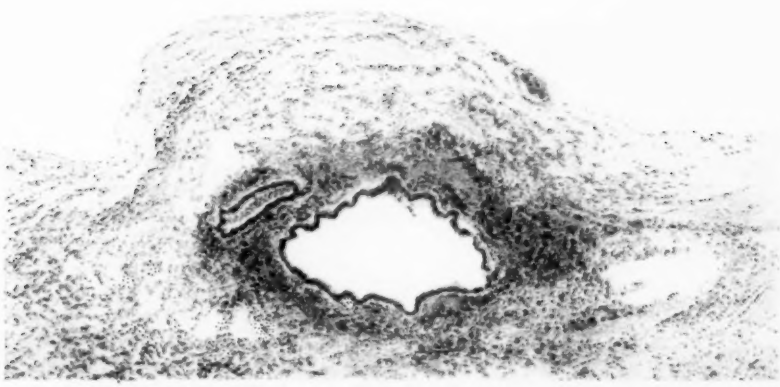

Fig. 23

PLATE I

and only three were over 6 cm. in diameter, the largest being about 9 cm. Perforation had occurred in many of the small ones a few millimeters in diameter, and in most of the hematomas over 1 cm. in diameter; but there was abundant opportunity to study hematomas of all sizes before perforation had taken place.

These hematomas arise from glands and tubules of endometrial (müllerian) type which penetrate the ovary from its surface, usually the lateral and the free surface. The hematomas may be superficial or deep. An ovary may contain only superficial hematomas or only deep ones, but often contains both, in various stages of development and retrogression. Perforation usually occurs in the superficial ones while they are still small, only a few millimeters in diameter. On the other hand the deep ones may reach a larger size, a few to several centimeters in diameter, before perforation occurs. It is impossible to group all of the specimens according to the situation of the hematomas, just as it is impossible to classify all uteri containing leiomyomas according to the situation of the latter. In both instances multiple growths are usually found in the periphery and the deeper portions of the ovary or the uterus. We may, however, study the development and retrogression of the superficial and deeper hematomas separately.

### THE DEVELOPMENT AND RETROGRESSION OF SUPERFICIAL HEMATOMAS (HEMORRHAGIC CYSTS) OF ENDOMETRIAL (MÜLLERIAN) TYPE

These hematomas arise from glands or tubules of endometrial (müllerian) type which are situated on the surface of the ovary or in its cortex. Hemorrhage occurs in the ovarian tissue about the gland, even when the lumen of the gland is small (Figs. 23 and 26). The lumen of the gland, or tubule, becomes larger and at first seems to be filled with serum giving rise to a small cyst with hemorrhage in its walls (Fig. 27). The blood surrounding the epithelial lining next ruptures through the overlying epithelial tissue carrying some of the latter with it and escapes into the cavity of the dilated gland or cyst (Figs. 28 and 29). In this way the ovarian hematoma arises. If the tension within the small hematoma is sufficiently great and the overlying wall of the cyst is thin, perforation occurs and some or all of the hemorrhagic contents containing some epithelium, cast off by menstruation, escapes into the peritoneal cavity (Fig. 30). I believe implantation adenoma of endometrial type may arise wherever this epithelium lodges on suitable soil whether on the surface of the ovary, about the site of the perforation, or on the peritoneal surface of the pelvic structures; especially in the culdesac. Perforation may occur in very small hematomas from 1 to 3 mm. in diameter. The perforation may become sealed and the hematoma reform, in time to

be followed by another or other perforations. If all of the epithelial lining is cast off by menstruation the life of the hemorrhagic cyst is ended, and all evidence of it may disappear. If most of the epithelium is removed and but a few cells persist, these cells may reform first into glands (Fig. 31) and later possibly into hematomas. It is also possible that all the epithelial lining may be cast off by the underlying hemorrhage rupturing into the lumen of the hematoma, and that the hemorrhagic cyst may die without perforation. The life of the hematoma or hemorrhagic cyst and its reaction to menstruation before and after perforation apparently depend on the presence of its epithelial lining.

The superficial hematomas are small, rarely over 5 mm. in diameter, more frequently between 1 and 3 mm. in diameter. If they have recently reacted to menstruation they are red in color, later bluish black (Figs. 21 and 22). They are usually multiple, and are often found in various stages of development and retrogression in the same specimen, suggesting that they are not all of the same age. The perforation of one may lead to the development of others by implantation on the surface of the ovary, or there may be multiple

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PLATE II

Fig. 24.—Plate II (Case 18). Reproduction of a colored photomicrograph (obj. 16 mm.) of a section of the peritoneal implantation adenoma shown in Fig. 17. Glands and tubules of endometrial type are present, the tissues of the peritoneum are thickened and recent stromal hemorrhage is situated in these tissues giving the "red raspberry" appearance of the implantation (Fig. 17). The histologic appearance of this implantation is similar to that shown in the preceding and the following illustration, except for the situation of the hemorrhage. The hemorrhage in the ovarian adenomas is more closely confined to the tissues surrounding the glands or tubules. As in the preceding instance, peritoneal implantations arise from epithelium escaping from the tube or other implantations (especially from the perforation of an ovarian hematoma). In this case I believe the epithelium came from or through the tube.

Fig. 25.—Plate II (Case 7). Reproduction of a colored photomicrograph (obj. 16 mm.) of a section through one of the small pigmented elevations on the lateral surface of the right ovary shown in Fig. 33. The gland-like spaces are lined by cuboidal epithelium; some of the cells are ciliated. There is an extravasation of blood in the tissues about the glands, which I interpret as a reaction to menstruation and believe that these glands arose from epithelium escaping from the tube or from the perforation of an ovarian hematoma as Fig. 30 from the same ovary. The operation occurred eighteen days after the last menstrual period and the patient menstruated every two to three weeks.

Fig. 26.—Plate II (Case 3).—Reproduction of a colored photomicrograph (obj. 16 mm.) of a section through a gland-like space imbedded in the superficial tissues of the right ovary (*b*. Fig. 41). The cells are cuboidal to columnar; some are ciliated. There is a marked extravasation of blood in the tissues about the gland which I interpret as a reaction to menstruation. The patient was operated upon the second day of the menstrual flow. It is from these gland-like structures that hematomas (hemorrhagic cysts) of endometrial type arise. This represents the first stage in the development of a superficial hemorrhagic cyst, namely, peripheral stromal hemorrhage.

Fig. 27.—Plate II (Case 7). Reproduction of a colored photomicrograph (obj. 16 mm.) through a dilated gland of endometrial type. The epithelial cells lining the gland are cuboidal to columnar, and many are ciliated. It is a miniature hemorrhagic cyst of endometrial type with hemorrhage about it. This represents the second stage in the development of a hemorrhagic cyst, namely, peripheral hemorrhage (subepithelial) and dilatation of the gland or tubule. The pigmented cells in the cavity of the cyst indicate that there has been a previous slight escape of blood through the epithelial lining.

Fig. 28.—Plate II (Case 5). Reproduction of a colored photomicrograph (obj. 16 mm.) through a section of one of the small pigmented elevations on the lateral surface of the right ovary shown in Fig. 2. The patient was menstruating the day of the operation. Subepithelial hemorrhage is present, and in one place the blood has ruptured through the epithelial lining and is escaping into the cavity of the miniature hemorrhagic cyst, carrying some of the epithelial cells with it. This represents the third stage in the development of a superficial ovarian hematoma, or hemorrhagic cyst of endometrial type, namely, hemorrhage into the cavity of the cyst. The wall of the cyst was torn in cutting the section.

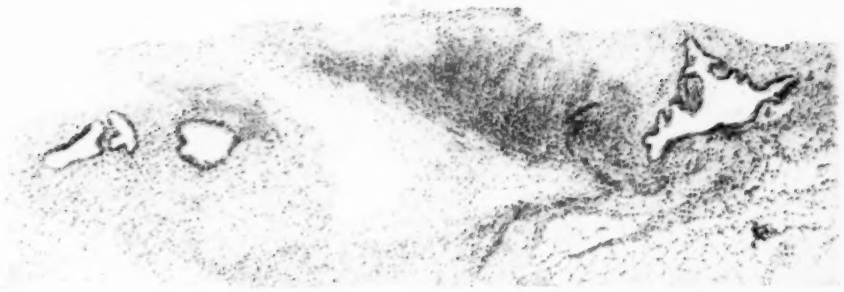

Fig. 24

Fig. 25

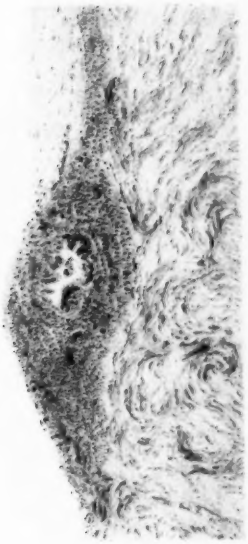

Fig. 26

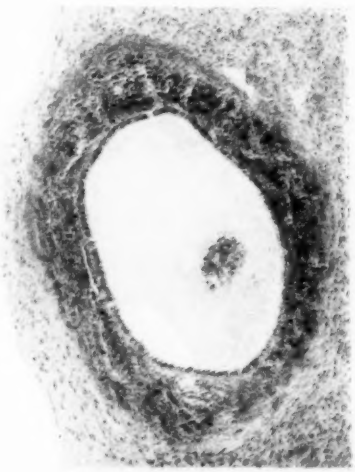

Fig. 27

Fig. 28

PLATE II

primary implantations from the tubes. They are most frequently found on the lateral and under surface of the ovary, very rarely on the mesial surface. The lateral and under surfaces of the ovary are the portions most frequently in contact with the fimbriae of the tube (Fig. 1).

### THE DEVELOPMENT AND RETROGRESSION OF DEEP OVARIAN HEMATOMAS (HEMORRHAGIC CYSTS) OF ENDOMETRIAL (MÜLLERIAN) TYPE

These arise from glands or tubules of müllerian type situated in the deep tissues of the ovary, which react to menstruation. It was impossible in every instance to determine the source of the tubules from which the hematomas arose, but in many specimens it was evident that the hematoma had developed from a gland or tubule which had invaded the ovarian tissue from its surface. Tubules were found in these specimens invading the ovarian tissue from its surface (Figs. 39 and 40) and also extending in various directions through the deeper tissues of the ovary. Hematomas developed in the dilated portions of these tubules (Fig. 54). While complete serial sections were not made of any of the ovaries, enough sections were made from some specimens to demonstrate that multiple small hematomas may arise in the tortuous course of a tubule, just as multiple ponds and lakes may arise in the course of a stream. Furthermore, even in some of the medium-sized and larger hematomas the tubule from which they apparently arose was found emptying into or growing away from them (Figs. 37 and 42). It is difficult to conceive why one portion of a tubule may develop into a hematoma while other portions of the same tubule fail to do so. The early stages in the development of the deeper hematomas are the same as the early stages in the development of the superficial hematomas. Three possible outcomes may result: First the hematoma, in time, reaches the surface of the ovary and perforation occurs; the hematoma may attain a large size (several centimeters in diameter) before this takes place. Secondly, the hematoma may increase in size so slowly that perforation never occurs and it ceases to grow after the menopause. Thirdly, the epithelial lining may be completely cast off in its repeated reactions to menstruation and the life of the hematoma ceases and if small, the remains may be completely absorbed.

The histologic study of the hematomas before perforation is most interesting. The reaction to menstruation manifests itself by hemorrhage into the ovarian tissues beneath the epithelial lining (Fig. 42); and by rupture through the overlying epithelium into the cavity of the hematoma, carrying with it some of the epithelial lining. This is followed by the attempt to repair, which manifests itself by two definite reactions. First, as there is no avenue for the escape of

the menstrual blood, other than the cavity of the hematoma or hemorrhagic cyst, some of it is retained in the wall of the cyst, disintegrates and is changed into blood pigment but the greater portion of it is retained in the cavity of the hematoma. Endothelial leucocytes may arise in the wall of the hematoma and wander out into its cavity through the breach caused by the menstrual hemorrhage (Figs. 47 and 50) and as phagocytes devour the red blood corpuscles (Fig. 45). As yet I am not convinced that they wander back into the ovarian stroma again carrying with them their load of disintegrated red blood corpuscles. The endothelial leucocytes both in the cavity of the hematoma and in the stroma break down, setting free the blood pigment. Hyaline fibrous tissue arises in the stroma of the denuded lining of the hematoma (Fig. 48). This process gives rise to a thickened pigmented lining of the hematoma without an epithelial covering which in certain stages of its development might be confused with the lining of a corpus luteum hematoma, the endothelial leucocytes being mistaken for luteal cells.

The second reaction in the attempt to repair, is the relining of the denuded portion of the wall of the hematoma by epithelium from epithelial cells which had not been cast off. This epithelium is found elongated and growing over the denuded pigmented lining of the hematoma (Fig. 51).

The histologic picture in different hematomas varies greatly. Some are completely or almost completely lined by epithelium with evidence of recent and old (blood pigment) hemorrhage in

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PLATE III

Fig. 29.—Plate III (Case 5). Reproduction of a colored photomicrograph (obj. 50 mm.) of a section through the pigmented elevation on the lateral surface of the left ovary, shown in Fig. 2. It is a small superficial hemorrhagic cyst of endometrial type. The greater portion of the epithelial lining has been cast off by the subepithelial hemorrhage (menstrual) rupturing into the cavity of the cyst. Epithelial and stroma cells are present in the small piece of tissue (*a*) shown in the hemorrhagic contents of the cyst (see also Fig. 32). Should the cyst rupture, the hemorrhagic contents, including epithelium and stroma cells, would escape into the peritoneal cavity. Should the epithelium fall on suitable soil, implantation adenoma would arise, which I have designated as secondary implantations, in contradistinction to the primary ones arising from the tube. Peritoneal implantations were not observed in this case, but I believe they would have arisen later from epithelium escaping through the perforation of the hemorrhagic cyst, had the cyst not been removed by operation.

Fig. 30.—Plate III (Case 7). Reproduction of a colored photomicrograph (obj. 50 mm.) through a superficial hemorrhagic cyst of endometrial type which had perforated. This is one of the pigmented elevations shown on the lateral surface of right ovary (Fig. 33). The walls of the cyst are collapsed and some of the epithelial lining has been cast off by the subepithelial hemorrhage in its walls rupturing into the cavity of the cyst. Some of this cast off epithelium was probably carried with the contents of the cyst, escaping through the perforation into the peritoneal cavity; it may have lodged on suitable "soil" and might have been the cause of some of the peritoneal implantation adenomas present in this case. Should all of the epithelium be cast off, the life of the cyst would probably cease. If a few cells were retained they might develop into adenomas, and give rise to the condition shown in the next illustration.

Fig. 31.—Plate III (Case 6 of second series[2]). Reproduction of a colored photomicrograph (obj. 50 mm.) through a small pigmented area on the lateral surface of the right ovary. The uterus contained multiple leiomyomas. Implantation adenomas of endometrial type were present in the culdesac, and also invading the appendix. Adhesions were present on the surface of the ovary. Two glands or tubules of endometrial type are shown, as well as yellow pigmented cells, the probable remains of hemorrhage in the ovarian stroma. These could be interpreted as the remains of a perforated hemorrhagic cyst in which nearly all of the epithelial lining had been cast off, and in the repair which followed, the epithelial cells which remained developed into glands. The implantation adenomas in this case might have arisen from the epithelium escaping through the perforation of the cyst.

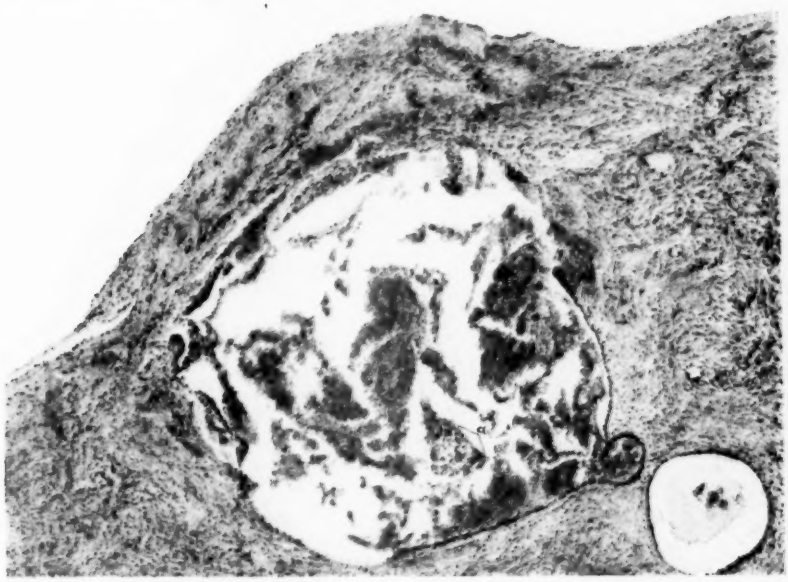

Fig. 29

Fig. 30

Fig. 31

PLATE III

the underlying tissues. In some the entire lining of the hematoma has reacted to menstruation while in others only small areas have reacted. In other hematomas the epithelial lining may be entirely or almost entirely destroyed and the hematoma is lined by the pigmented tissue described above. In many of the specimens one finds several or all of the stages in the reaction of the hematoma to menstruation and the attempted repair following it. In places there is an epithelial lining without the underlying hemorrhage, in other situations evidence of the hemorrhage rupturing into the cavity with loss of the overlying epithelium, endothelial leucocytes forming and escaping into the cavity, the development of the pigmented layer from the endothelial leucocytes, the breaking down of the endothelial leucocytes and deposit of blood pigment in the tissues of this layer, the formation of hyaline connective tissue and finally the

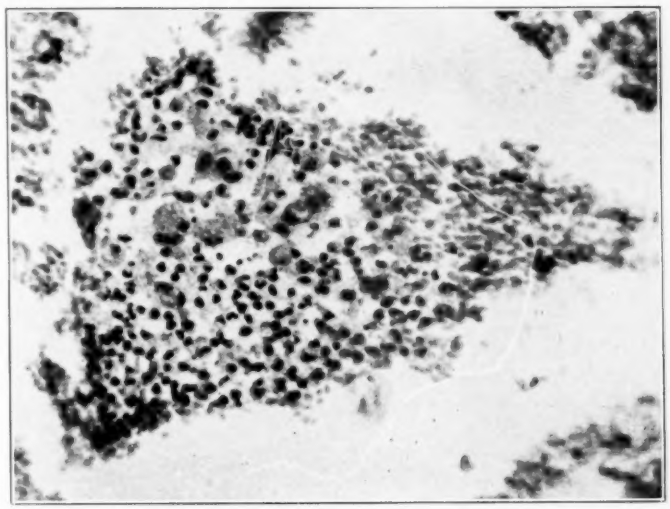

Fig. 32.—(Case 5). Photomicrograph (obj. 4 mm.) of a section of the piece of tissue in the hemorrhagic contents of the hematoma shown in Fig. 29. It consists of epithelial cells, stroma cells and leucocytes, surrounded by red blood corpuscles. Had perforation occurred prior to the operation this piece of tissue would have escaped into the peritoneal cavity, and might have developed into an implantation adenoma of endometrial type. Possibly the "nature" of the blood (menstrual), as well as the leucocytes and stroma cells, may irritate the peritoneum and facilitate the implantation of the epithelium. Clinical observations have shown that the material escaping from these cysts differs from normal blood in that it is very irritating and causes adhesions.

attempt to reline the denuded area from epithelium which has not been cast off by menstruation. Usually all parts of the hematoma do not react to menstruation at the same time. The process of repair is apparently slow and all of the epithelium may be destroyed. The life of the hemorrhagic cyst apparently ceases with the destruction of its epithelial lining (Fig. 56). Some of these hemorrhagic cysts or hematomas lose all of their epithelial lining and "die" before they reach the surface of the ovary and perforate.

The mechanism of perforation can be readily explained by menstrual hemorrhage in a thin portion of the wall of the cyst, which

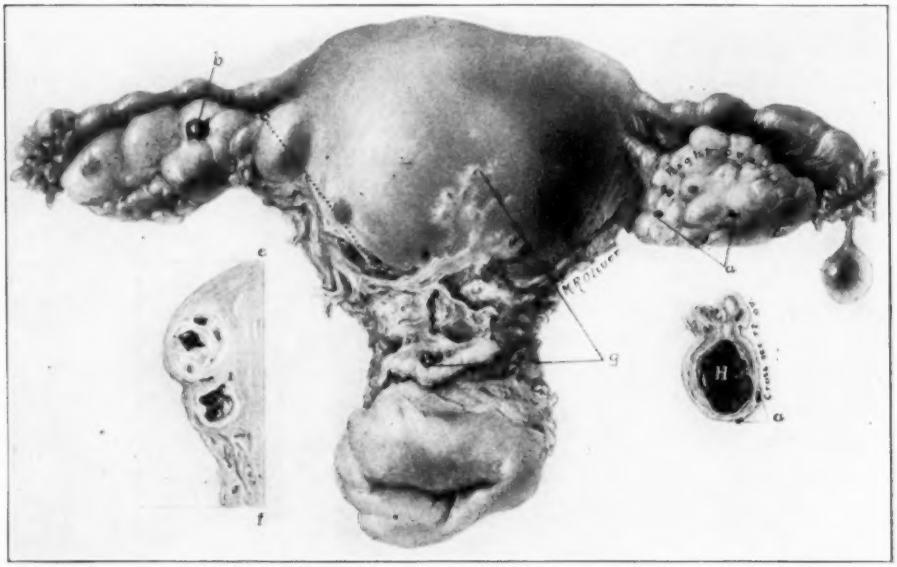


Fig. 33.—(Case 7). Superficial hematomas of endometrial type of both ovaries in various stages of development (some had perforated); implantation adenoma of endometrial type invading the posterior surface of the uterus and forming an "adenomyoma" of that organ; leiomyomas of the uterus, uterine polyps. Posterior view of the specimen removed at operation, ovaries turned upwards exposing their lateral surfaces (x 3/5). The superficial hematomas (*a*) of the right ovary appear as pigmented elevations (photomicrographs of two of these were shown in Figs. 25 and 30). A typical corpus luteum hematoma (*H*) is shown in the cross section of the right ovary. Implantation adenoma is superficially invading the posterior surface of the uterus in the area marked by the pointers (*g*). An "adenomyoma" shown in the insert (*e* - *f*) was present in the uterine wall, indicated by the line (*c* - *d*). I believe that the implantation adenoma invading the wall of the uterus, as well as the "adenomyoma" arose from epithelium implanted on the surface of the uterus which escaped either from the tubes (both were patent) or from the perforating hematoma of the ovary, or probably from both. A photomicrograph of a section of a portion of the "adenomyoma" is shown in the next illustration.

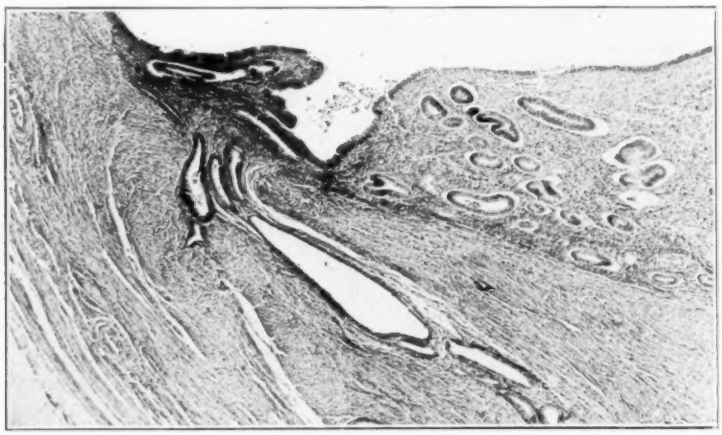

Fig. 34.—Photomicrograph (obj. 50 mm.) of a section of a portion of the "adenomyoma" shown in the insert (*e* - *f*) of Fig. 33. As stated in the previous illustration I believe that this "adenomyoma" arose from epithelium implanted on the surface of the uterus, escaping either from the tubes or from the perforation of a small superficial hematoma of the ovary as shown in Fig. 30.

has reached the surface of the ovary. The hemorrhage ruptures first either into the peritoneal cavity or into the cavity of the cyst; per-

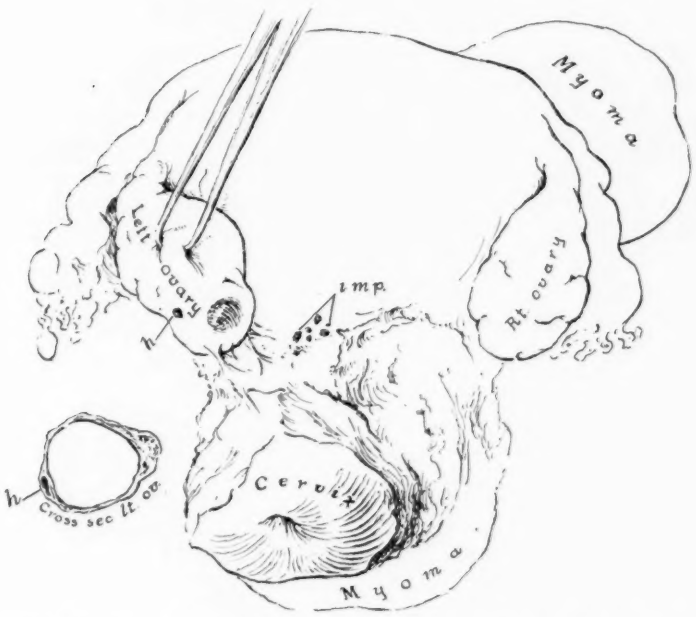


Fig. 35.—(Case 10). Remains of a superficial hematoma of the left ovary, implantation adenoma on the posterior surface of the uterus, multiple leiomyomas of the uterus. Posterior view of the specimen removed at operation (x 4/5). The left ovary was lightly adherent to the posterior surface of the uterus. It is turned upwards, exposing the lateral surface and showing the pigmented elevation (*h*); this is also indicated in the cross section of the cystic ovary. Histologically this pigmented elevation is due to a small cyst-like cavity filled with old blood and without an epithelial lining. I believe that it represents a small hemorrhagic cyst of endometrial type in which the epithelial lining had been cast off. The implantation adenoma (imp.) involving the posterior surface of the uterus may have arisen from epithelium escaping from the tubes (both were patent), or from epithelium escaping from the ovarian hematoma which might have perforated.

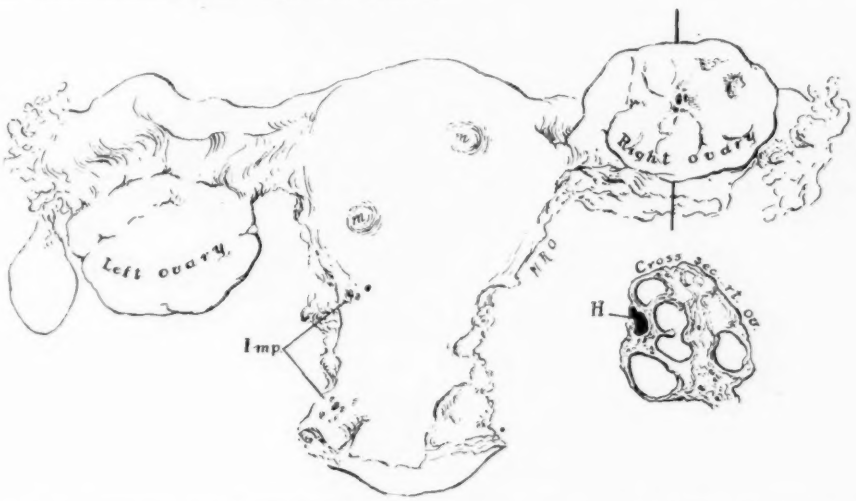


Fig. 36.—(Case 11). Hemorrhagic cyst (hematoma) of endometrial type of the right ovary; implantation adenoma (imp.) of the posterior surface of the uterus and both utero sacral ligaments (right one not shown in the illustration), multiple small leiomyomas of the uterus. Posterior view of the specimen removed at operation, right ovary turned upwards showing the pigmented elevation on its lateral surface; the hematoma (*H*) is also shown in the cross section of the ovary (x 4/5). The hematoma of the ovary might have perforated, and the implantations involving the posterior surface of the uterus and the uterosacral ligaments might have arisen from epithelium escaping from it, or from epithelium escaping from the tubes (both were patent), or from both. I believe the ovarian hematoma (Fig. 37) arose from epithelium escaping from the tubes.

foration occurs and some of the contents of the cyst escapes into the peritoneal cavity carrying with it epithelial cells which apparently give rise to implantation adenomas wherever the epithelium falls on suitable "soil." The escape of the contents of the cyst probably prolongs its life. It relieves tension and thus favors repair until the next reaction to menstruation. There is evidence that the perforation may close if it is small without the ovary or cyst becoming adherent to some other adjacent structure (Fig. 60). In the majority of cases the ovary or cyst becomes adherent to some other pelvic tissue such as the side of the pelvis, the posterior layer of the broad ligament, or the uterine wall; and these assist in sealing the perforation. In freeing the ovary or hematoma at operation the

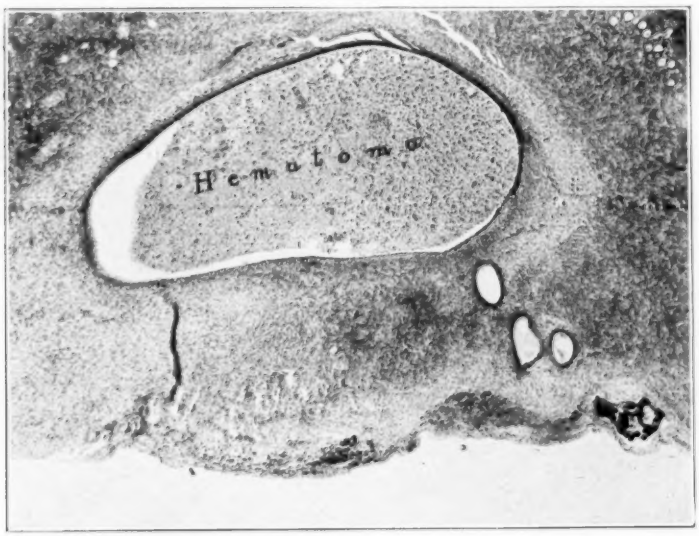


Fig. 37.—(Case 11). Photomicrograph (obj. 50 mm.) of a section of the hematoma of the right ovary (Fig. 36). The hematoma is lined by cuboidal epithelium, some of which were ciliated. A tubule of endometrial type is shown extending towards the hematoma from the surface of the ovary. I believe the hematoma arose from this tubule and the tubule from epithelium escaping from the tube and becoming implanted on the surface of the ovary; both tubes were patent. Other sections showed that the hematoma had probably perforated, as indicated in the cross section of the ovary shown in Fig. 36.

perforation is usually reopened or the healed thin wall is torn at this place as was demonstrated in the first communication.[1] The future life history of the perforated cyst is similar to that of the cyst before perforation; another or repeated perforations may occur, the menopause may take place before another perforation, or the epithelial lining may be completely destroyed and further reaction to menstruation cease. The perforation, in all cases which I have studied, has been situated on the lateral surface or free border of the ovary and never on its mesial surface. This would indicate that the hematomas arose from tubules which had invaded the ovarian tissues

from these surfaces, the portions of the ovary which are normally most frequently in contact with the fimbriated end of the tube (Fig. 1).

The contents of the hematomas vary, consisting of blood (menstrual), blood pigment, cast off epithelium, endothelial leucocytes, cholesterin crystals, and stroma cells, in various stages of activity and disintegration, and in various proportions, depending upon the age of the hematoma and upon the phase of the menstrual cycle in which the hematoma was removed. An ovarian hematoma may not react to

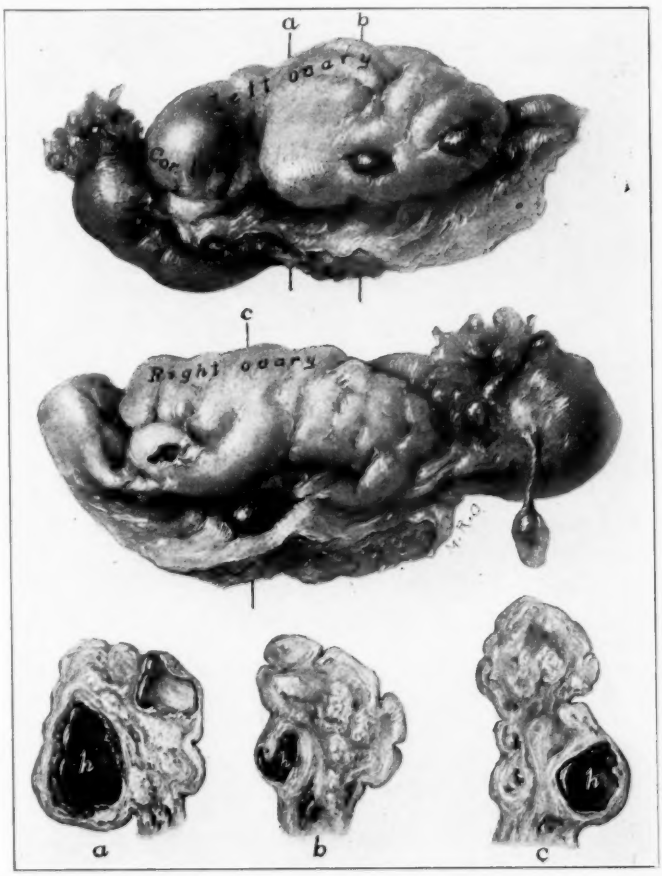


Fig. 38.—(Case 8). Multiple hemorrhagic cysts of endometrial type of both ovaries without gross evidence of perforation; small implantation adenoma in the culdesac (see Fig. 59); multiple leiomyomas of the uterus, uterine polyp. Lateral surface of both ovaries and cross sections of the ovaries showing the situations of three of the hematomas (x 1). A corpus luteum hematoma was present in the distal pole of the left ovary. Cross section (*a*) through the plane (*a*) of the left ovary shows a hematoma (*h*) of endometrial type, and cross section (*b*) through the plane (*b*) shows a smaller hematoma. A photomicrograph of the latter appears in Fig. 42. (*c*) shows a hematoma of endometrial type in the right ovary. All the hematomas were presenting towards the lateral surfaces of the ovaries, the surface which is most frequently found in contact with the fimbriated end of the tube. Both tubes were patent.

every menstrual period. The recuperation (regeneration) after menstruation is apparently slow and if the damage is great as the result of one menstrual period it may not be sufficiently recuperated to take part in the next one.

THE INFLUENCE OF PREGNANCY ON OVARIAN HEMATOMAS (HEMORRHAGIC CYSTS) OF ENDOMETRIAL (MÜLLERIAN) TYPE

As these cysts react to menstruation causing the formation of a hematoma, we would expect their epithelial lining also to react to pregnancy, just as the uterine mucosa reacts to it. The opportunities

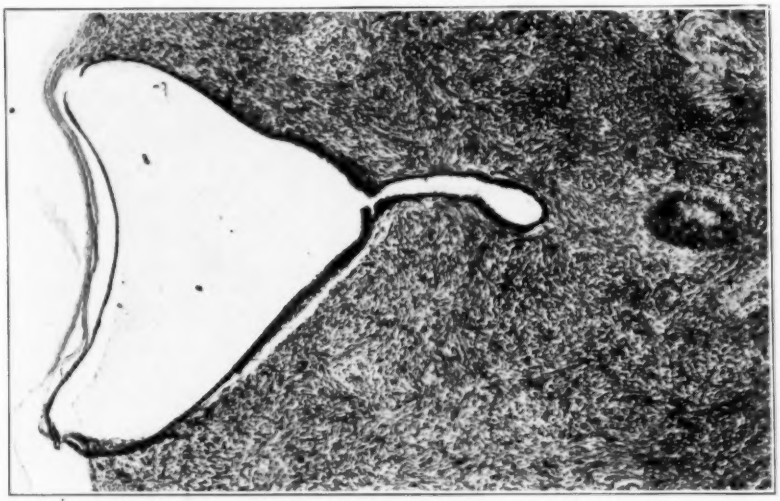

Fig. 39.—(Case 8). Photomicrograph (obj. 16 mm.) of a section of a tubule of endometrial type invading the tissues of the ovary from its lateral surface (left ovary, Fig. 38). Some of the epithelial cells were ciliated.

to study this reaction are few for several reasons. Patients with this condition rarely become pregnant, due to the ovarian lesion which is often bilateral, and to other conditions frequently associated with it which are conducive to sterility, as leiomyomas and polyps and

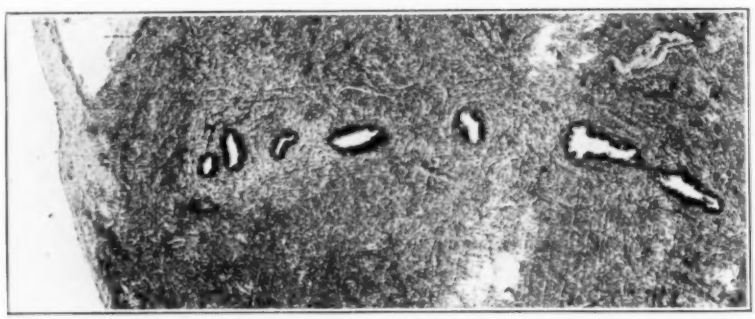

Fig. 40.—(Case 8). Photomicrograph (obj. 16 mm.) of a tubule of endometrial type invading the deeper tissues of the ovary from its lateral surface (left ovary, Fig. 38). Some of the epithelial cells were ciliated. It is from these tubules that the deeper hematomas arise, and they may reach a large size before perforation occurs.

the age of the patient, the latter part of the child bearing period. Sterility or no pregnancies in several years in married women with these ovarian hematomas is quite a common condition. Should a patient with this lesion become pregnant and the latter condition be

recognized, there would probably not be sufficient indications present to justify an abdominal operation.

I have observed only one instance of pregnancy associated with an ovarian hematoma of endometrial type. Both conditions were accidental findings (Case 14 of this series). The patient, aged 37, had been married for eleven years and had never been pregnant. Menstruation had been regular, the last flow occurring two weeks before the operation. At the operation, which was undertaken for multiple leiomyomas of the uterus, the left ovary was found to be adherent to the posterior surface of the broad ligament; in freeing it the characteristic "chocolate" like fluid escaped. The entire uterus, both tubes and ovaries and appendix were removed. An apparent implantation adenoma was present on the posterior uterine wall ad-

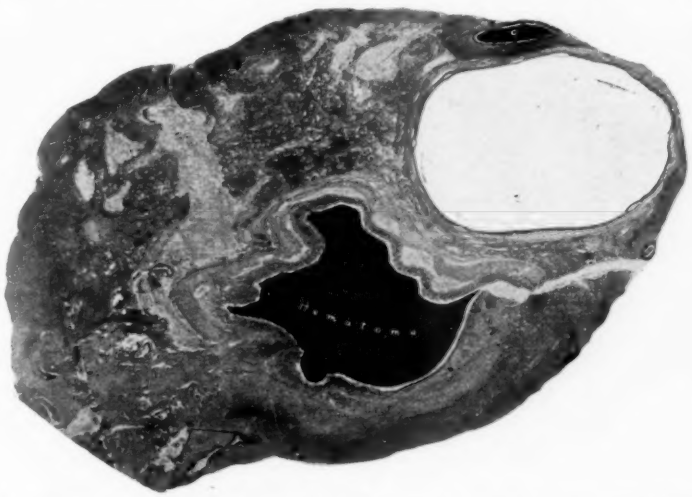

Fig. 41.—(Case 3). Cross section of the right ovary showing a hematoma (*H*) of endometrial type which had not perforated (x 5). The ovary was incised before it had been hardened, and some of its contents escaped; hence the walls of the hematoma are partially collapsed. The lining of the hematoma showed various stages in the reaction to menstruation; the attempt to absorb the menstrual blood, and to reline the denuded portions from epithelium which had not been cast off by menstruation (see Figs. 45, 46, 47, 48 and 50). Tissue of endometrial type was found invading the ovary at (*a*) from its lateral surface, and possibly the hematoma developed from a tubule arising from this tissue, which had penetrated the deep structures of the ovary. Serial sections were not made, and thus this point was not determined. Only about one quarter of the circumference of the hematoma was lined by epithelium. "*b*" indicates the gland with hemorrhage about it shown in Fig. 26, and "*c*" a small hemorrhagic cyst lined by ciliated epithelium.

jacent to the attachment of the left broad ligament and the site of the perforation of the ovarian hematoma (Fig. 67). The gross appearance of this implantation adenoma differed from the usual ones in the absence of menstrual pigmentation. On opening the uterine cavity an early pregnancy was found, the embryo being 14 mm. long (Fig. 68). Both fallopian tubes were apparently normal and the corpus luteum of pregnancy was situated in the right ovary. The hematoma of the left ovary was lined by typical decidual tissue, with the surface epithelium still present in the depressions (Figs. 69 and

70). Histologically it was identical with that of the compact layer in the decidua vera of the pregnant uterus. Glands were not present in the lining of the ovarian hematoma. There was likewise a decidual reaction in the implantation adenoma in the posterior uterine wall. One of the dilated tubules resembled a miniature uterine cavity (Fig. 71), a portion of whose mucosa showed a definite compact and spongy layer. The reaction in the compact layer was not quite as striking as that in the wall of the ovarian hematoma, but the glands of the spongy layer had a histological structure identical with that of the glands in the spongy layer of the decidua vera of the uterine cavity (Fig. 72). Sections from three different portions of both fallopian tubes were examined microscopically and no decidual reaction was found in any.

The typical decidual reaction of pregnancy was found in the lining of cavities in three different situations of this specimen, the cavity of the uterus, the ovarian hematoma and those of the implantation adenoma of the posterior uterine wall. The uterine cavity was lined by endometrium. What was the nature of the lining of the other two? I believe it was similar to that of the uterine cavity.

### THE INFLUENCE OF THE MENOPAUSE AND OLD AGE ON OVARIAN HEMATOMAS (HEMORRHAGIC CYSTS) OF ENDOMETRIAL (MÜLLERIAN) TYPE

As the reaction of the lining of these hematomas to menstruation and pregnancy is similar to that of the endometrium, we should expect that the menopause and old age would affect the mucosa of these hematomas, just as they affect the mucosa of the uterine cavity. I believe that this is true. In many instances the reaction of the mucosa of the hemorrhagic cysts to menstruation completely removes the epithelial lining and causes the death of the cyst before the menopause occurs. If small, the remains of the hematoma may be completely absorbed, if large, it may persist possibly for the life of the individual, the old "menstrual" blood being retained in a cyst-like cavity lined by a deeply pigmented membrane (Fig. 56).

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### PLATE IV

Fig. 42.—Plate IV (Case 8). Reproduction of a colored photomicrograph (obj. 50 mm.) of a section of the hematoma of endometrial type appearing in the cross section of the ovary shown in (*b*) Fig. 38. Most of the epithelial lining had been cast off by menstruation. In places it is present with an underlying hemorrhage. In one place a typical "uterine" gland (*g*) is present in this stroma. Cross sections are shown of what appears to be a tubule (*t*) which may have invaded the ovary from its lateral surface, and may have been the source of the hematoma (see also Figs. 39 and 40 from the same ovary). Some of the epithelial cells lining the hematoma were ciliated. Should perforation occur the contents of the hematoma, including epithelium cast off by menstruation, would escape into the peritoneal cavity; the latter might give rise to implantation adenomas.

Fig. 43.—Plate IV (Case 4 of second series[2]). Reproduction of a colored photomicrograph (obj. 4 mm.) of a portion of the wall of the ovarian hematoma shown in Fig. 62. Here the hematoma is lined by columnar epithelium (not ciliated). A cross section of a dilated thin walled blood vessel is present, with the red blood corpuscles escaping through its wall into the surrounding stroma; indicating the source of the subepithelial hemorrhage. In other sections the epithelial lining had been cast off by the underlying hemorrhage rupturing into the cavity of the hematoma.

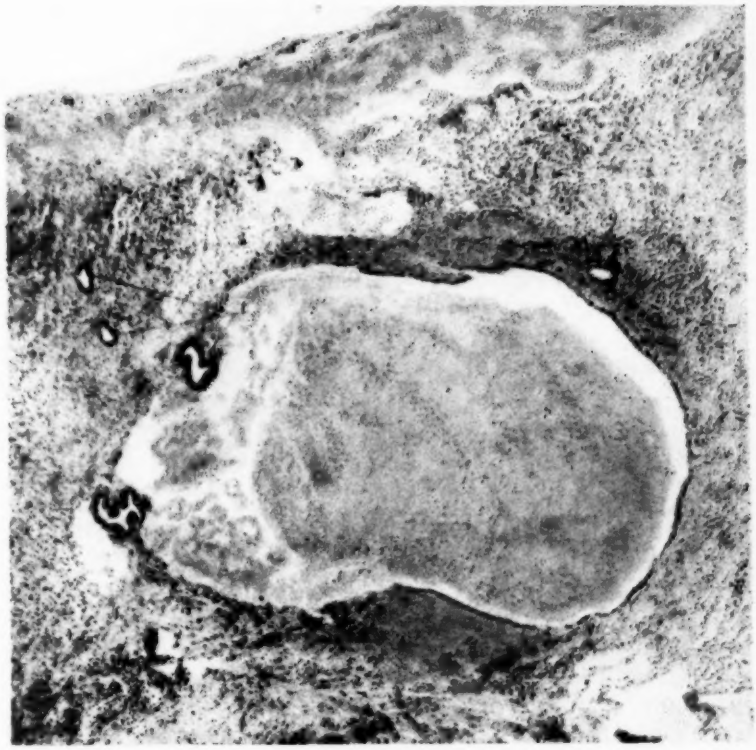

Fig. 42

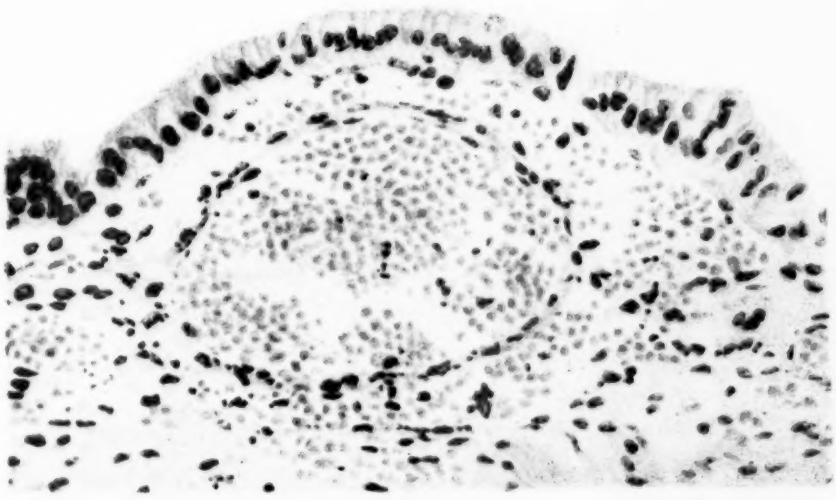

Fig. 43

PLATE IV

I have had the opportunity to study only two cases with ovarian hematomas of endometrial type in women past the menopause. The first one occurred in a woman 61 years old (Case 6 of this series). The remains of a hematoma (Fig. 73) were found in the right ovary containing "caked" menstrual blood in a cavity lined by a wavy hyaline membrane deeply pigmented with hemosiderin. The epithelial lining had completely disappeared (Fig. 74); this had possibly occurred before the menopause. Implantation adenoma was present invading the posterior uterine wall (Fig. 75), which histologically resembled the mucosa of the uterine cavity.

The second case occurred in a woman 59 years old with carcinoma of the body of the uterus. Small ovarian hematomas of endometrial type were present in both ovaries. The epithelial lining was present in both hematomas. There was no histologic evidence of any recent reaction to menstruation. The specimen was only recently removed and I have not had an opportunity to complete its study and am not reporting it in full in this series of cases.

### THE EVIDENCE THAT THESE HEMATOMAS OR HEMORRHAGIC CYSTS ARE OF ENDOMETRIAL (MÜLLERIAN) TYPE

They develop from glands or tubules in the ovary which are lined by cuboidal or columnar epithelium (often ciliated) resembling tubal and uterine epithelium. Hemorrhage occurs in the ovarian tissue about the glands or tubules at the time of menstruation.

The glands or tubules from which these hematomas develop are derived from epithelium which has escaped from the fimbriae, or through the lumen of the fallopian tube, and becoming implanted on the surface of the ovary has invaded its tissues. See text of this article for evidence supporting this view.

Histologically, the epithelial lining of these ovarian hematomas is similar to that of the hematomas and dilated cavities found in primary adenomyoma of the uterus and of the tube.

Every variation in the histologic structure of the lining of the ovarian hematomas (often seen in different portions of the same hematoma) is due to different phases in its reaction to menstruation, such as: the inactive stage without evidence of hemorrhage, the subepithelial hemorrhage, the escape of blood through the overlying epithelium carrying some of the latter with it into the cavity of the hematoma, the subsequent reaction on the part of the ovarian stroma lining the hematoma in its attempt to absorb the extravasated blood, and especially the development and activities of the endothelial leucocytes as scavengers, and finally the regeneration of the epithelial lining over the denuded portions from epithelium which had not been removed by menstruation. The perforation of the hematoma is but a result of menstruation.

Their reaction to menstruation, pregnancy and old age is similar to that of the mucosa of the uterine cavity. They develop during the menstrual life of women, and the condition found in portions of the lining of the hematoma may correspond to the phase of the menstrual cycle indicated by the menstrual history of the patient. In the only case of pregnancy associated with this condition which I have studied, the decidual reaction in the lining of the ovarian hematoma was histologically identical with that of the compact layer of the decidua vera of the accompanying uterus.

The histologic study of these hematomas shows that portions of the epithelial lining are cast off by the menstrual hemorrhage, and this epithelium may be found free in the hemorrhagic contents of the hematoma. Where perforation has occurred adenoma of endometrial type may be found on the surface of the ovary about the site of the perforation and in the other tissues adherent or adjacent to it, as well as in situations where material escaping from such a perforation would be likely to lodge. These implantation adenomas are apparently derived from material escaping from the perforation of the ovarian hematoma, and in structure are of endometrial type; often resembling normal endometrium more closely than the lining of the ovarian hematoma. If adenoma of endometrial type develops from the implantation of epithelium cast off by menstruation from the lining of the hematoma and escaping through the perforation, then the epithelial lining of the hematoma must also be of endometrial type.

## REPORT OF CASES

In the first series of cases[1] reported by me, an ovarian hematoma or hemorrhagic cyst with evidence of perforation was present in each instance. The second series of cases[2] included those with implantation adenoma of endometrial type involving some portion of the intestinal

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### PLATE V

Fig. 44.—Plate V (Case 9). Reproduction of a colored photomicrograph (obj. 4 mm.) of a portion of the wall of the hematoma shown in Fig. 60, which is here lined by epithelium. The epithelium for the most part is low, but in a small depression it is cuboidal and ciliated. What appears to be a dilated (blood) vessel filled with endothelial leucocytes is present beneath the epithelial lining.

Fig. 45.—Plate V (Case 3). Reproduction of a colored photomicrograph (obj. 4 mm.) of a portion of the wall of ovarian hematoma shown in Fig. 41. This portion had not reacted to menstruation. The cells are cuboidal to columnar and ciliated; in places they are "heaped up" as is often found in glandular hypertrophy of the uterine mucosa, and also in the ampulla of the fallopian tube. The contents of the hematoma is shown above the epithelial lining. It consists for the most part of endothelial leucocytes engorged with red blood corpuscles and also of red blood corpuscles in various stages of preservation. For the source of the endothelial leucocytes see the next two illustrations.

Fig. 46.—Plate V (Case 3). Reproduction of a colored camera lucida drawing of a microscopic section of a portion of the wall of the hematoma shown in Fig. 41; it is reacting to menstruation (a little lower magnification than the preceding illustration). The patient was operated upon the second day of the menstrual period. The epithelium to the left is ciliated; to the right the cilia have disappeared, and the epithelium has assumed various shapes as though undergoing retrogression. Subepithelial hemorrhage is present and endothelial leucocytes have appeared, some of which are filled with red blood corpuscles. A thin-walled blood vessel is shown in longitudinal section.

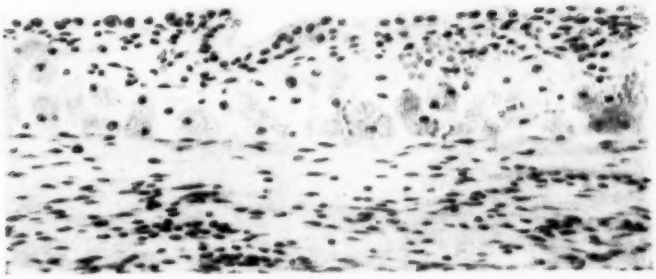

Fig. 44

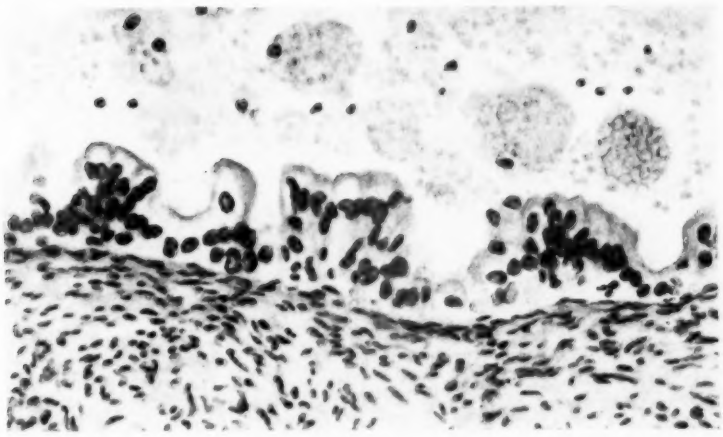

Fig. 45

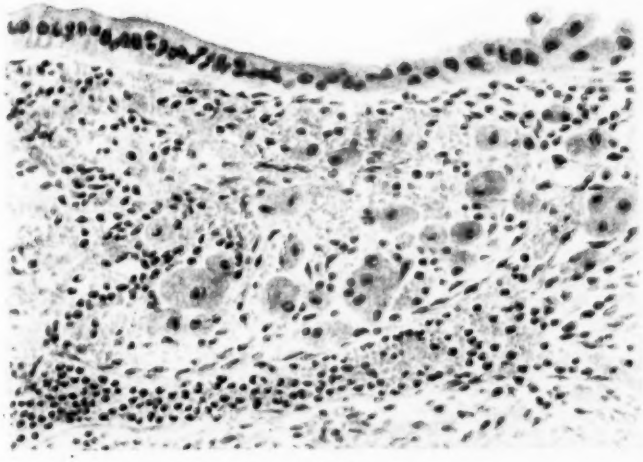

Fig. 46

PLATE V

tract. The cases in these two series represent, for the most part, those of long standing—namely the advanced stages of the disease.

The cases reported in the present series were chosen from those demonstrating the origin and development of ovarian hematomas of endometrial type and ovarian hematomas without perforation rather than from those with perforation and extensive implantations. A few additional cases are included which showed special features in the life history of this condition. The cases in this series represent, for the most part, the early stages of the disease, and as a whole are not of as great clinical interest as those reported in the other two series.

CASE 1.—(Case 7 of the first series).[1] *Multiple hematomas (of endometrial type) of both ovaries (some had perforated), adhesions between the ovaries and the posterior uterine wall (implantation adenoma not found), small leiomyomas of the uterus.* Mrs. X. W., aged thirty-five, complained of uterine bleeding which had occurred six times in the last twelve weeks. She had never been pregnant. Menstruation had been regular, moderate and normal until the last three months. Since then it had been more profuse and frequent and had been accompanied with pain. Pelvic examination showed the uterus to be slightly enlarged, irregular in consistency, with an "inflammatory" mass on either side. The preoperative diagnosis was a myomatous uterus with bilateral "chronic pelvic peritonitis." Operation was at the Albany Hospital, Nov. 10, 1918. Both ovaries were cystic and adherent to the posterior surface of the uterus and broad ligaments. Small leiomyomas were present. On freeing the ovaries a small amount of chocolate fluid escaped. The appendix, both tubes and ovaries and the entire uterus were removed (Fig. 52). The tubes appeared normal. Both ovaries contained multiple hematomas in various stages of development and retrogression; most of them had not perforated. At the time of the previous communication[1] I believed that some of these hematomas represented various stages in the life cycle of corpus luteal hematomas. I have restudied this specimen and with my present knowledge recognize that all of the hematomas represent various stages in the life history of hematomas of endometrial type. Many sections were studied from both ovaries (nearly all the tissue in both ovaries has been examined histologically). Adenoma of endometrial type (Fig. 53) was found on the surface of both ovaries, which invaded the ovarian tissue in the form of tubules. These tubules were found throughout the entire substance of both ovaries and one could trace the development of the hematomas from these tubules (see Figs. 54 and 55). The patient made a satisfactory convalescence.

CASE 2.—*Hematoma (of endometrial type) of the right ovary without evidence of perforation or implantations in the pelvis.* Mrs. W. N., aged twenty-eight, complained of sterility and painful menstruation. She had been married seven years and had never been pregnant. Menstruation began at the age of thirteen and had always been painful. The pain began with the flow and was worse the second day. It had increased in severity the last year and was more marked on the right side. The last flow occurred four weeks before the operation. Pelvic examination showed that the uterus was small, in normal position and freely movable. The right ovary seemed enlarged and very tender. The preoperative diagnosis was a cystic right ovary. Operation was at the Albany Hospital July 31, 1920. The right ovary was cystic, about twice the normal size, and slightly adherent. Otherwise the pelvic organs appeared normal. The cyst (retention) of the right ovary was excised, and in doing this a small hematoma about 5 mm. in diameter was noticed near the lateral surface of the ovary; this was also excised. The appendix had been removed

ten years ago. The cervix was dilated, and a stem pessary introduced. The small hematoma was lined in part by cuboidal epithelium. The rest of the hematoma was lined by a pigmented layer of ovarian tissue composed for the most part of endothelial leucocytes. Histologically the lining of the hematoma was similar to that of other hematomas of endometrial type described in this communication. The patient made a satisfactory convalescence.

CASE 3.—*Hematomas (of endometrial type) of both ovaries, without evidence of perforation or implantations in the pelvis, uterine polyp, multiple leiomyomas and descensus of the uterus, weakened pelvic floor.* Mrs. C. L., aged thirty-six, complained of descensus of the uterus and profuse menstruation. She had one child nine years old and one miscarriage a year later. Menstruation had been prolonged and profuse for the last year. The patient was menstruating the day of the operation. Pelvic examination showed a weakened pelvic floor, uterus irregularly enlarged and in descensus. The preoperative diagnosis was a weakened pelvic floor, descensus of the uterus and multiple small leiomyomas.

Operation was at the Albany Hospital March 22, 1921. The uterus was found to be irregularly enlarged by multiple small leiomyomas. The left ovary was of normal size and contained a reddish pigmented elevation about 4 mm. in diameter on its lateral surface. A similar but smaller elevation was present on the mesial and also on the lateral surface of the right ovary near its free border. Adhesions were not present in the pelvis nor was there any gross evidence of any implantations. (The pelvis was examined very carefully for both.) The appendix, both tubes and ovaries and the body of the uterus were removed. The cervix was sutured into the abdominal incision and the pelvic floor was repaired. The uterus contained several small leiomyomas, the largest being 3.5 cm. in diameter. A small polyp was also present in the fundus of the uterus. The left ovary contained on its lateral surface a superficial hematoma about 4 mm. in diameter which was lined by low to cuboidal epithelium; many of the epithelial cells were ciliated. Three similar superficial hematomas were present in the right ovary, the smallest being about 0.5 mm. (Fig. 26) and the largest 4 mm. in diameter. A larger, deep hematoma, about 1 cm. in diameter was situated in the right ovary which had not ruptured but was presenting towards its lateral surface (Fig. 41). This hematoma was partly lined by epithelium which in places was ciliated; subepithelial stromal hemorrhages were also present (Figs. 45 and 46). The greater portion of this hematoma was lined by a pigmented layer of ovarian tissue without an epithelial covering, the pigment, the result of previous hemorrhage, being for the most part in cells of the type of endothelial leucocytes (Figs. 47 and 48). The superficial hematomas could have arisen

## PLATE VI

Fig. 47.—Plate VI (Case 3). Reproduction of a colored photomicrograph (obj. 16 mm.) of a portion of the wall of the hematoma (*H*) Fig. 41, showing endothelial leucocytes escaping into the cavity of the hematoma, and the formation of the pigmented lining. To the right, the epithelial lining is still present with an underlying stromal hemorrhage. In the center, endothelial leucocytes have developed and are pouring into the cavity of the hematoma through a breach; probably caused by a previous subepithelial hemorrhage rupturing into the cavity of the hematoma, and carrying the overlying epithelium with it (see also Fig. 50.). To the left, the luteal-like pigmented layer is forming which is composed, for the most part, of endothelial leucocytes containing blood pigment. The contents of the cavity of the hematoma (above) consists of blood and endothelial leucocytes, epithelium and stroma cells in various stages of preservation.

Fig. 48.—Plate VI (Case 3). Reproduction of a colored photomicrograph (obj. 16 mm.) of a portion of the wall of the hematoma (*H*) Fig. 41, showing the pigmented layer lining the greater portion of the hematoma. This represents a later stage of the condition shown in the preceding illustration. The endothelial leucocytes are breaking down, and hyaline connective tissue has developed in the stroma of the wall of the hematoma.

Fig. 49.—Plate VI (Case 9). Reproduction of a colored photomicrograph (obj. 16 mm.) of a portion of the wall of the ovarian hematoma shown in Fig. 60. This demonstrates very well the breaking down of the endothelial leucocytes in the superficial portion of the wall of the hematoma, and the deposit of blood pigment in the deeper tissues.

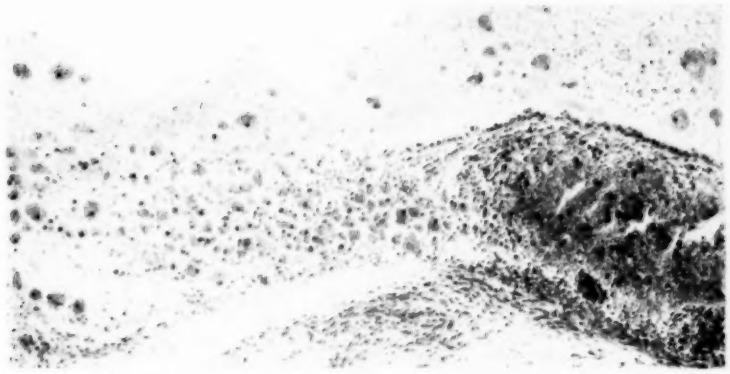

Fig. 47

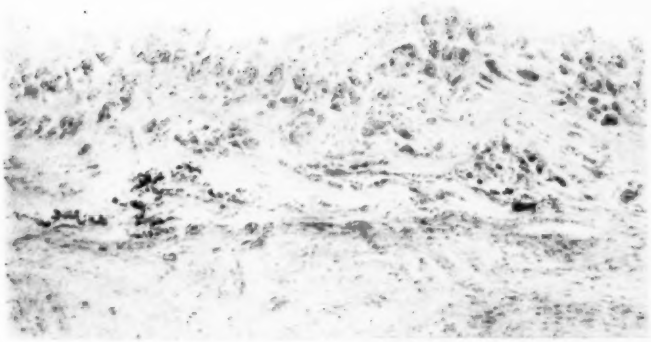

Fig. 48

Fig. 49

PLATE VI

The Matthews Northrup Works, Buffalo

from implantations of epithelium derived from or through the tube, while the deeper one could have arisen from tubules penetrating the ovary from its surface. Perforation or rupture of these hematomas might give rise to implantation adenoma wherever epithelium carried with the material escaping into the pelvis might fall on suitable "soil." The patient made a satisfactory convalescence.

CASE 4.—*Multiple small implantation adenomas (of endometrial type) of the posterior surface of the uterus, posterior culdesac, anterior surface of the left broad ligament and in the anterior culdesac, leiomyoma of the uterus, evidence of stromal hemorrhage in the right ovary.* Mrs. D. B., aged forty-six, complained of uterine bleeding. She had been married for eleven years and had never become pregnant. Menstruation had been regular, normal and without pain. For the last two months there had been a more or less constant bloody uterine discharge, at times quite profuse. Pelvic examination showed that the uterus was irregularly enlarged and

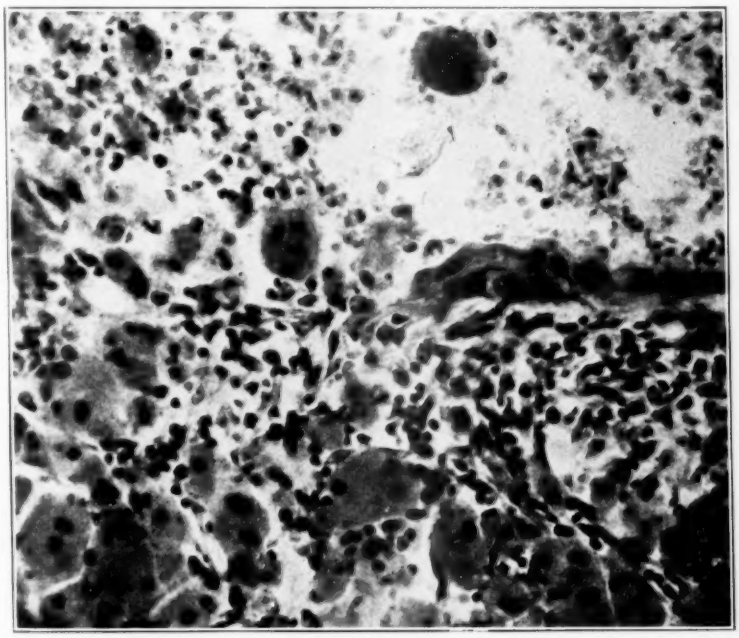

Fig. 50.—(Case 3). Photomicrograph (obj. 4 mm.) of a portion of the wall of the ovarian hematoma shown in the center of Fig. 47. The endothelial leucocytes are escaping into the cavity of the hematoma through a breach in the overlying epithelium; probably caused by a previous hemorrhage (menstrual).

freely movable. The preoperative diagnosis was uterine leiomyoma and possibly carcinoma of the body of the uterus.

Operation was at the Albany Hospital May 6, 1921. Implantation adenoma was found as described above (Fig. 10). The appendix, both tubes and ovaries and the entire uterus were removed. Multiple small leiomyomas were found, the largest being 3 cm. in diameter. Carcinoma of the body of the uterus was not present. The implantations were of endometrial type. A small pigmented area 3 mm. in diameter was present on the lateral surface of the right ovary. Histologically this pigmentation was due to old blood pigment in the cells of the ovarian stroma; glands or tubules of endometrial type were found neither in this area, nor in any of the sections taken from both ovaries. This area may represent the remains of a small ovarian hematoma of endometrial type in which all of the epithelium had

been destroyed by menstruation and perforation. The tubes appeared normal, and there were not any pelvic adhesions present. I believe the implantations probably arose from epithelium escaping from the tubes, although the possibility of implantations from a small ovarian hematoma which had disappeared after perforation cannot be excluded. The patient made a satisfactory convalescence.

CASE 5.—*Superficial small hematomas (of endometrial type) of both ovaries without gross evidence of perforation and of implantation adenomas in the pelvis, retroflexed uterus, appendicitis with abscess.* Mrs. J. C., aged twenty-three, complained of attacks of pain in the right side of the abdomen. She had not had any children but had had one miscarriage a year and a half ago. Menstruation was moderate in amount, and had always been painful. The patient was menstruating the day of the operation. The first attack of pain in the right side of the abdomen occurred five months before her operation; she was in bed a week at that time. The last attack occurred twelve days before the operation; she was in bed five days, had nausea, vomiting and elevation of temperature. Abdominal palpation showed

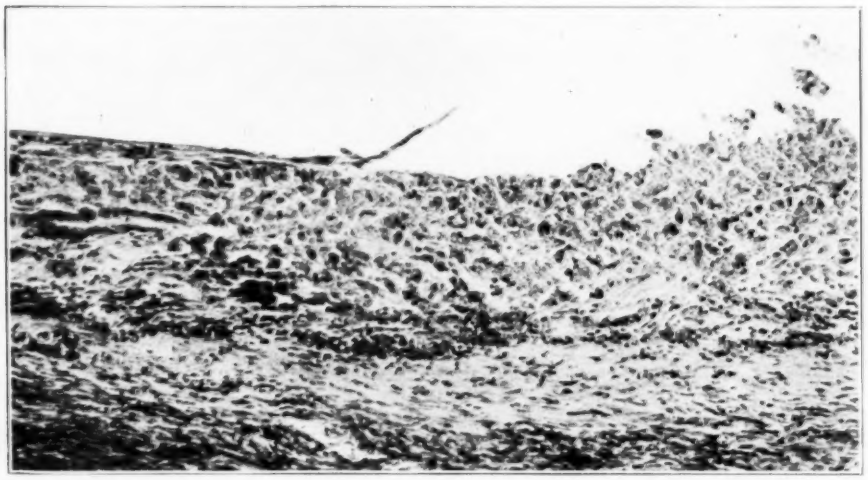

Fig. 51.—(Case 9). Photomicrograph (obj. 16 mm.) of a portion of the wall of the ovarian hematoma present in Fig. 60, showing the flattened epithelium to the left "riding" over the pigmented layer, which is composed mostly of endothelial leucocytes undergoing retrogression. This represents the relining of the denuded portion of the hematoma, from epithelium not cast off by menstruation (from the same section shown in Fig. 44). The retrogression of the endothelial leucocytes is well demonstrated in Fig. 49; also from the same specimen but another section.

marked tenderness and resistance over the region of the cecum. Pelvic examination showed a retroflexed uterus. The preoperative diagnosis was appendicitis and retroflexion of the uterus. Operation was at the Albany Hospital May 9, 1921. A median incision was made; the uterus was retroflexed. On replacing it and examining the ovaries, multiple superficial hematomas from 1 to 2 mm. in diameter were noticed on the lateral surface of the right ovary and a similar hematoma about 3 mm. in diameter on the lateral surface of the left ovary (Fig. 2). The right tube and ovary were removed; the portion of the left ovary containing the hematomas was excised. The uterus was suspended by suturing the round ligaments to its posterior surface. A small abscess was found about the appendix lateral to the cecum. The appendix was removed and the seat of the abscess was drained by a protective wick drain through a small McBurney incision. No gross evidence of implantation adenomas was found in the pelvis. Histologically, the small ovarian hematomas were of endometrial type with a definite reaction to menstruation, which had car-

ried some of the overlying epithelium into the cavities of the hematomas (Figs. 28 and 29). Should perforation occur some of this epithelium would escape with the contents of the hematoma into the pelvis, and might give rise to implantation adenomas. Both tubes were patent and the hematomas on the surface of the ovaries probably arose from epithelium escaping from the tubes. The patient made a satisfactory convalescence.

CASE 6.—*Hematoma of endometrial type (perforated) of the right ovary, implantation adenoma (of endometrial type) of the posterior uterine wall, multiple small leiomyomas, uterine polyp, adherent retroflexed uterus.* Mrs. L. B., aged sixty-one, complained of indigestion and loss in weight. She had four children, the youngest being thirty-two years old. Menopause occurred at the age of forty-eight

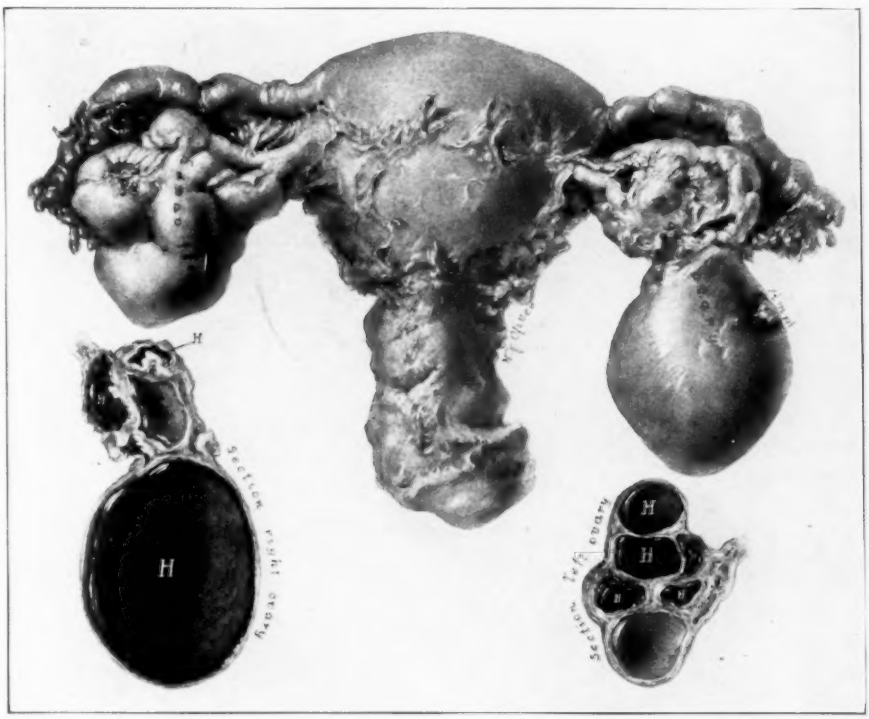


Fig. 52.—(Case 1). Multiple hematomas of endometrial type, in various stages of development and retrogression, of both ovaries. Posterior view of the uterus, tubes and ovaries removed at operation (x 2/3); both ovaries also shown in cross section. The hematomas are marked "H" in the cross sections of the ovaries and a follicular cyst "C" is shown in each ovary. All the hematomas were of endometrial type. Implantation adenoma of endometrial type was found on the surface of both ovaries, invading the underlying tissues (Fig. 53); tubules of endometrial type were found in the deeper tissues of the ovary and hematomas arose from these just as the superficial hematomas developed (Fig. 54 compare with Fig. 37). As the hematomas became larger the typical pigmented lining developed in the portion of the wall where the epithelium had been cast off by menstruation. In some of the hematomas all of the epithelium had been cast off, and the hematoma was completely lined by the pigmented layer; and I believe that the life of the hemorrhagic cyst had ceased, and eventually the remains might be absorbed (Figs. 55 and 56). In some of the hematomas quite typical "endometrial" tissue was present (Fig. 57), and in others tubules were found communicating with the lining of the hematoma, and extending into or from the ovarian tissue (Fig. 58). Both tubes were patent, and while adenoma was found on the surface of both ovaries, none was found on the posterior surface of the uterus.

and was normal in character. She had lost forty pounds in weight in the last two years without any definite symptoms other than slight indigestion and constipation ("always constipated"). X-rays of the gastrointestinal tract were negative. Ab-

dominal palpation was also negative. Pelvic examination showed an adherent retroflexed uterus with marked induration in the culdesac. Rectal palpation confirmed the above and I remarked that if the patient had not passed the menopause I would make a diagnosis of implantation adenoma of endometrial type in the culde-

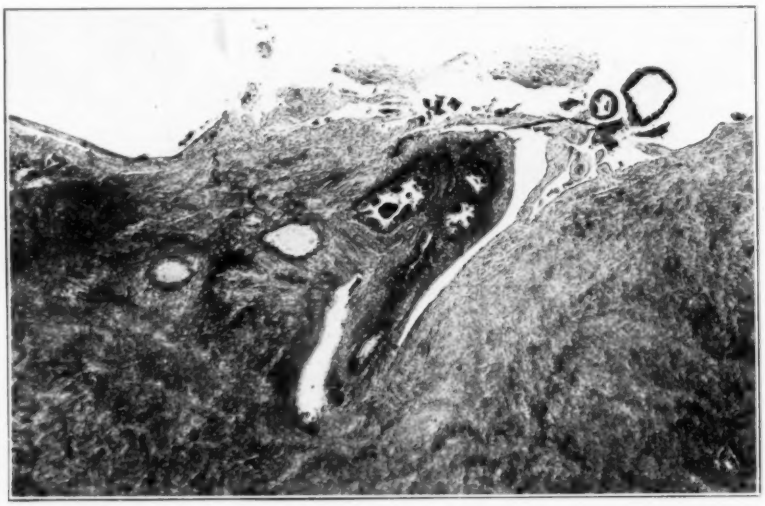

Fig. 53.—(Case 1). Photomicrograph (obj. 50 mm.) showing adenoma of endometrial type on the surface of the ovary and invading the underlying tissue as tubules.

sac. The preoperative diagnosis was probably carcinoma (primary source not determined) with implantations in the culdesac. An exploratory operation was suggested but it was not urged as I did not believe that it would be of any great benefit to the patient. Operation was at the Albany Hospital June 22, 1921. A median ab-

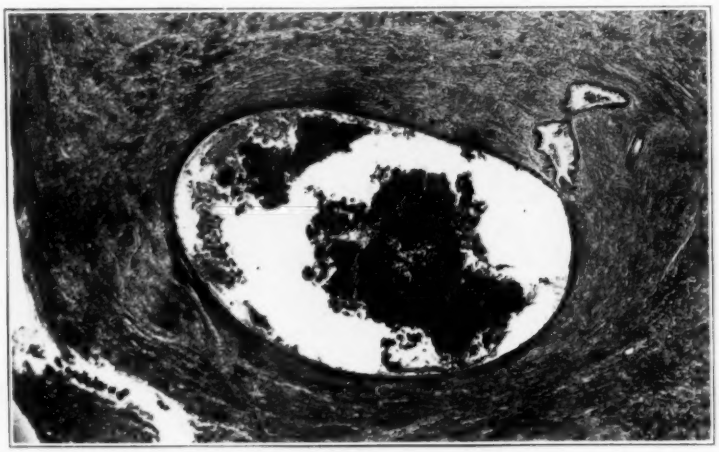

Fig. 54.—(Case 1). Photomicrograph (obj. 50 mm.) showing a small hematoma which has arisen from a dilated portion of a tubule, similar to the tubules invading the ovary from the adenoma on the surface (Fig. 53 compare with Fig. 37).

dominal incision was made and the contents of the abdominal cavity were carefully palpated and nothing abnormal was detected. The appendix was removed. The uterus was found to be retroflexed and densely adherent to the bottom of the culde-

sac. The left tube and ovary were normal. The right tube appeared normal but the right ovary was cystic, about 4 cm. in diameter, and densely adherent to the side of the pelvis. On freeing the ovary the walls of the cyst were torn, and thick "chocolate-like" paste escaped with hard "caked" pieces of material similar to the paste. The entire uterus and both tubes and ovaries were removed. The uterus contained multiple small leiomyomas, a polyp, and adenoma of endometrial type of the posterior uterine wall, which had apparently invaded the uterus from its peritoneal surface (Figs. 73 and 74). Histologically the glandular elements of this adenoma were similar to that of the mucosa lining the uterine cavity. The wall of the ovarian hematoma was thin. Many sections were made from different portions of the wall of the hematoma. Every section showed a similar condition; a stroma consisting of wavy hyaline bands of tissue without cellular elements, and scattered through it old blood pigment (Fig. 75, compare with Figs. 48 and 49). An epithelial lining was not found. I believe that the ovarian hematoma represents the remains of a larger hematoma of endometrial type in which the epithelial lining had been destroyed and the hemorrhagic contents had never been completely absorbed. The

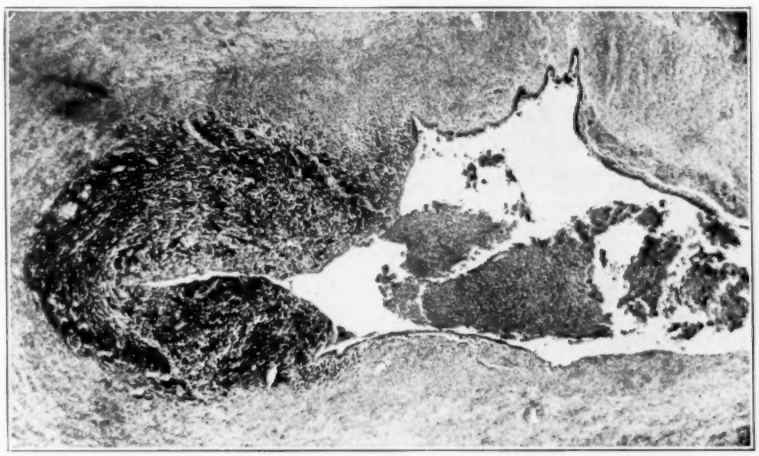

Fig. 55.—(Case 1). Photomicrograph (obj. 50 mm.) of a section of a small hematoma of endometrial type. To the left the epithelial lining is replaced by a thick pigmented layer, the result of previous hemorrhage. The rest of the hematoma, shown in this section, is lined by epithelium mostly cuboidal. In places subepithelial hemorrhage is present.

patient made a satisfactory convalescence and feels much better than before the operation.

CASE 7.—*Multiple small superficial hematomas (of endometrial type) of both ovaries with evidence of perforation, corpus luteum hematoma of the right ovary; implantation adenoma (of endometrial type) involving the posterior wall of the uterus and the culdesac, leiomyomas, uterine polyps, weakened pelvic floor and gallstones.* Mrs. I. B., aged forty-three, complained of uterine bleeding, indigestion and a sense of lack of support. She had one child twenty years old, the only pregnancy. Menstruation had recently been very profuse and irregular, occurring every two to three weeks, and was not accompanied by any pain. The last menstrual flow occurred eighteen days ago and she expected to menstruate any day. Occasionally there was slight bleeding independent of menstruation. She was not constipated. Pelvic examination showed a weakened pelvic floor, the uterus to be retroverted and slightly enlarged, with some induration in the culdesac. The preoperative diagnosis was a weakened pelvic floor, leiomyoma of the uterus, and possible implantation adenoma in the culdesac.

Operation was at the Albany Hospital June 30, 1921. The appendix, both tubes and ovaries and the entire uterus were removed. The gall bladder (gallstones) was excised and the pelvic floor was repaired. The left ovary contained two hematomas of endometrial type, 2 and 4 mm. in diameter, presenting on the lateral surface of the ovary near its free border. The larger one of these had possibly perforated. The right ovary contained several superficial hematomas of endometrial type from 1 to 2 mm. in diameter, all presenting on its lateral surface (Fig. 33). Some of these had perforated (Fig. 30). Cilia were present on some of the epithelial cells lining these hematomas. A typical corpus luteum hematoma 1.3 cm. in diameter was present in the right ovary. Adenoma of endometrial type was found invading the posterior wall of the uterus, especially marked near the left ovary (Figs. 33 and 34). Similar adenoma was also found in the culdesac. What is the relation between the hematomas of the ovaries and the adenomas invading the posterior wall

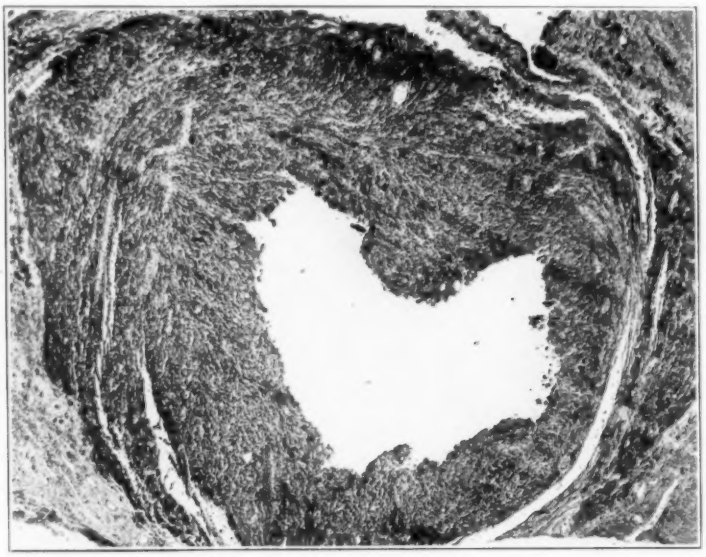

Fig. 56.—(Case 1). Photomicrograph (obj. 50 mm.) of a section of the remains of a hematoma of endometrial type, in which all of the epithelial lining has been cast off and is replaced by a thick pigmented wall similar to that shown in Fig. 55. I believe that the life of the hemorrhagic cyst, as such, had ceased.

of the uterus and the culdesac? Epithelial cells escaping from the perforated ovarian hematomas could have given rise to other implantations on the surface of the ovaries, and to the adenoma in the culdesac and uterine wall. They all could have had a common origin from epithelium escaping from the tubes. I believe that in this case implantations occurred from both sources. The primary implantations occurred from the tubes which were patent with free fimbriae. The perforating ovarian hematomas from time to time would expel into the pelvis, epithelium which might lodge on the surface of the ovaries or the posterior surface of the uterus, or in the culdesac. Implantations in various stages of development were found in all these situations. The uterine cavity contained multiple polyps. The patient died on the third day with symptoms of a severe infection and beginning pneumonia of the right lung but no signs of peritonitis. An autopsy was refused but immediately after death a blood culture was obtained by inserting a long hypodermic needle into the heart. A pure culture of pneumococcus Type II was obtained.

CASE 8.—*Multiple hematomas (of endometrial type) of both ovaries without gross evidence of perforation, small implantation adenoma (of endometrial type) in the culdesac, multiple leiomyomas, retroflexion of the uterus, weakened pelvic floor and hemorrhoids.* Mrs. M. L. H., aged fifty-one, complained of a sense of lack of support, indigestion and hemorrhoids. She had had one child eighteen years ago. Menstruation was regular, without pain and usually profuse in amount. The last flow occurred two weeks before the operation. Pelvic examination showed a weakened pelvic floor and the uterus to be irregularly enlarged and retroflexed. The preoperative diagnosis was multiple leiomyomas in a retroflexed uterus, and a weakened pelvic floor. At the operation at the Albany Hospital, Sept. 21, 1921, the uterus was found to be irregularly enlarged and freely movable. Both ovaries contained multiple small hematomas, but were not adherent. A small, thickened area, with a pigmented elevation about 2 mm. in diameter was detected in the culdesac (Fig. 59). This was excised and proved to be an adenoma of endometrial type. The appendix, both tubes and ovaries and the entire uterus were removed. The pelvic floor was repaired and the hemorrhoids were removed by the clamp and cautery.

Fig. 57.—(Case 1). Photomicrograph (obj. 16 mm.) of a section of a portion of the walls of one of the larger hematomas of endometrial type, shown in the cross section of the left ovary (Fig. 52). Histologically it resembles uterine mucosa.

Tubules of endometrial type were found invading both ovaries from their lateral and free surfaces. Three hematomas of endometrial type were present in the left ovary, the largest being about 1.7 cm. in diameter and the smallest about 5 mm. in diameter. Two of the hematomas apparently had not perforated, but one possibly had (Fig. 38). A corpus luteum hematoma was also present in the left ovary. All three hematomas were presenting on the lateral surface of the ovary. The right ovary contained two hematomas also unruptured, the largest being about 1 cm. in diameter. The uterus contained multiple leiomyomas, the largest being about 4 cm. in diameter. For the description of the ovarian hematomas see Figs. 38, 39, 40 and 42, with their legends. The tubules of endometrial type in the ovaries from which the hematomas had developed apparently arose from epithelium invading the ovaries from their surface, and could have been implanted from epithelium arising from or escaping through the fimbriated extremity of the tube. The implantation in the culdesac could have arisen from epithelium escaping from a small perforation in one of the ovarian hematomas (Fig. 42), or could have arisen from epithelium escaping from the tube, just as I believe the ovarian hematomas arose. Both tubes were apparently normal and patent. I believe that perforation of the hematomas would be followed by further implantations. The patient made a satisfactory convalescence.

CASE 9.—*Hematoma (of endometrial type) of the left ovary with healed perforation, implantation adenoma of endometrial type on the posterior surface of the right broad ligament at its uterine attachment, small leiomyomas and early carcinoma of the uterine cervix.* Mrs. G. S., aged forty-two, complained of uterine bleeding. She had two children, thirteen and nine years of age respectively. Menstruation was regular, quite profuse and occasionally accompanied with slight pain. She had been bleeding for five weeks prior to the operation. Pelvic examination showed that the cervix was lacerated and bled slightly on palpation; the uterus was enlarged and firm in consistency. The preoperative diagnosis was probably myofibrosis of the uterus. Preliminary curettage was advised to exclude the possibility of an early carcinoma of the uterine cervix. The uterus was curetted and a small piece of the cervix was excised under nitrous oxide anesthesia. The material removed did not suggest carcinoma in its gross appearance, but histologically typical squamous cell carcinoma was found in one fragment of the tissue

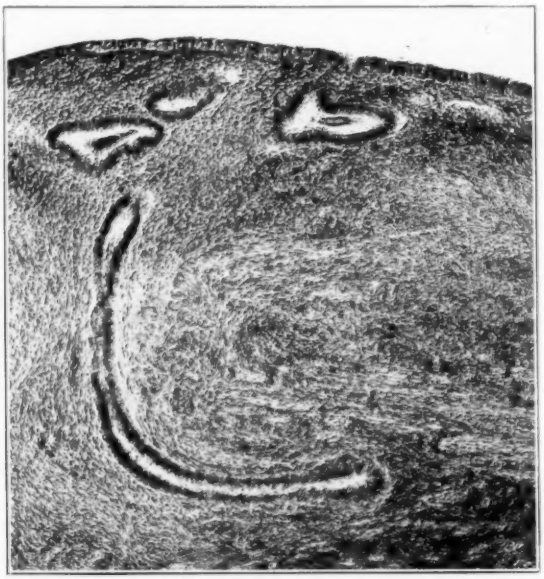

Fig. 58. —(Case 1). Photomicrograph (obj. 16 mm.) of a section of a portion of the wall of one of the larger hematomas of endometrial type, shown in the cross section of the left ovary (Fig. 52) (not from the hematoma shown in the preceding illustration). This portion of the hematoma is lined by tissue resembling uterine mucosa. A tubule is shown in longitudinal section, apparently invading the underlying ovarian tissue from the lining of the hematoma; or possibly the hematoma developed from this tubule. Less than one-third of the hematoma was lined by epithelium. The greater portion of the hematoma was lined by a thick pigmented wall similar to that shown in the previous illustrations.

removed. The patient was persuaded, with difficulty, to consent to a radical operation. On October 20, 1921, at the Albany Hospital, the entire uterus, left tube, and ovary and appendix were removed. The patient was insistent that some ovarian tissue should be saved if possible and the right tube and ovary, which appeared normal, were not removed. The left ovary, which was lightly adherent to the posterior surface of the broad ligament, contained a hematoma of endometrial type, about 2.5 cm. in diameter, which had apparently perforated on its lateral surface (Fig. 60). Adenoma of endometrial type was also present on the surface of the ovary, about the healed perforation (probably implantations from the perforation). Tubules of endometrial type were present in the ovarian tissue near the

attachment of its lateral surface to the broad ligament. Implantation adenoma of endometrial type was present on the posterior surface of the left broad ligament, at its uterine attachment (Fig. 60). The ovarian hematoma could have arisen from epithelium escaping from the tube. The implantation on the surface of the broad ligament could have arisen from the tube, or through the perforation of the ovarian hematoma adjacent to it. I believe more likely the latter. The patient made a satisfactory convalescence.

CASE 10.—*Small hematoma of endometrial type of the left ovary (perforated?), implantation adenoma of the posterior wall of the uterus, multiple leiomyomas uterine polyp.* Miss E. T., aged forty-one, complained of prolonged, profuse menstruation. Menstruation had been prolonged and profuse for three years, sometimes lasting two or three weeks. It was not accompanied by any pain. The last flow occurred three weeks before the operation. Pelvic examination showed the uterus to be irregularly enlarged and freely movable. The preoperative diagnosis was multiple leiomyomas of the uterus. At the operation at the Albany Hospital,

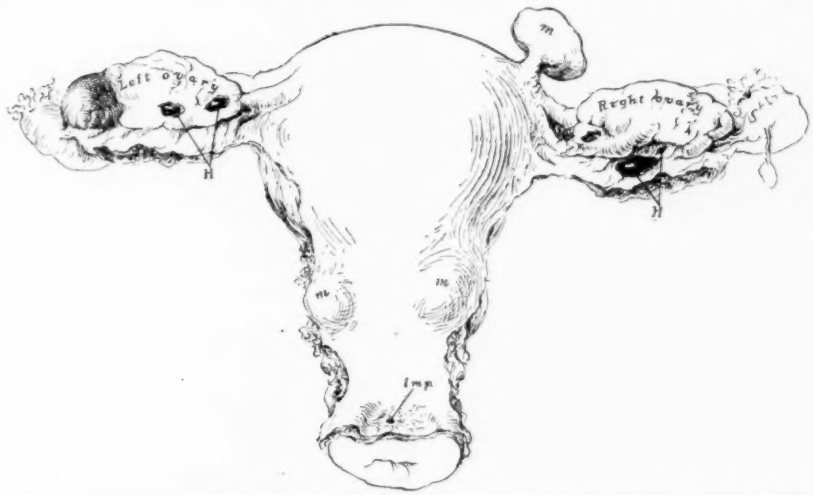


Fig. 59.—(Case 8). Multiple hematomas of endometrial type, in various stages of development, of both ovaries without gross evidence of perforation. Small implantation adenoma of endometrial type in the culdesac. Posterior view of the specimen removed at operation (x 1/2); both ovaries turned upwards exposing their lateral surfaces, and showing the hematomas presenting on this surface. I believe that the ovarian hematomas arose from epithelium escaping from the tubes, becoming implanted on the lateral surfaces of the ovaries and invading the underlying tissues of tubules (See Figs. 1, 38, 39, 40 and 42). The adenoma in the culdesac (*imp.*) may also have arisen from epithelium escaping from the tubes, or from epithelium escaping through the perforation of a small ovarian hematoma which was overlooked. The perforation of the ovarian hematomas would probably be followed by implantation adenoma wherever the contents of the hematoma lodged on suitable soil (compare with Figs. 60, 61 and 62 where perforation has occurred).

November 9, 1921, the uterus was found to be irregularly enlarged by multiple leiomyomas, the largest myoma being about 4 cm. in diameter. The entire uterus and both tubes and ovaries were removed. (The appendix had been removed at a previous operation.) The left ovary was cystic, of normal size, and lightly adherent to the posterior surface of the uterus (Fig. 35). There was a small, pigmented elevation about 3 mm. in diameter on the lateral surface of the ovary, which histologically proved to be a cyst-like cavity filled with pigmented material, the remains of an old hemorrhage, and without an epithelial lining. I believe it represents a small cyst of endometrial type in which the epithelial lining has been destroyed. Implantation adenoma of endometrial type was found invading the

posterior surface of the uterus lateral to the left ovary. Both tubes were apparently normal and patent. Two possible sources suggest themselves for the origin of the adenoma on the posterior surface of the uterus—one, the ovarian hematoma which had perforated (adhesions about the ovary), and in which later the perforation had become closed, and the other from epithelium escaping from the tube. The patient made a satisfactory convalescence.

CASE 11.—*Small superficial hematoma (of endometrial type) of the right ovary (perforated?); implantation adenoma of the posterior surface of the uterus and the utero-sacral ligaments; multiple small leiomyomas, retroversion of the uterus.* Mrs. N. M., aged twenty-nine, complained of very severe dysmenorrhea. She had been married twelve years, and had never been pregnant. Menstruation began at twelve

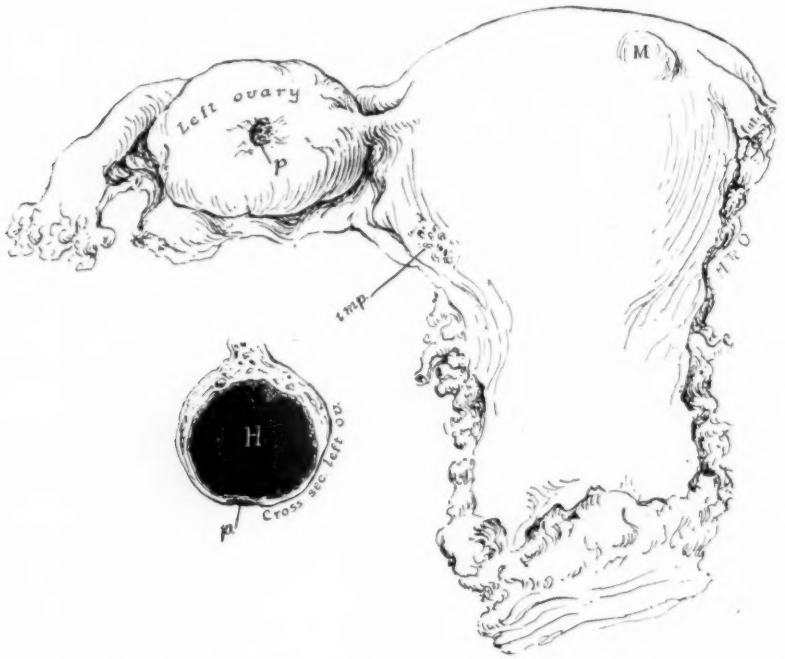


Fig. 60.—(Case 9). Hematoma of endometrial type of the left ovary with evidence of small perforation, implantation adenoma of endometrial type (imp.) on the posterior surface of the left broad ligament, near the perforation of the ovarian hematoma; early cancer of the uterine cervix. Posterior view of the specimen removed at operation (x 3/4); left ovary turned upwards, exposing the perforation (*p*), on the under surface of the ovary, which had healed over. Photomicrographs of portions of the wall of the ovarian hematoma are shown in Figs. 44, 49 and 51. Adenoma was present on the surface of the ovary about the perforation, and also on the posterior surface of the broad ligament (imp.) adjacent to it. I believe that all of these could have resulted from epithelium escaping through the perforation of the hematoma, although their origin from epithelium escaping from the tubes cannot be excluded.

years of age, always painful but more so the last three years, and increasing in severity. Menstruation lasted two to three days and was scanty in amount. Pain was present before the flow began and lasted during the entire menstrual period. The last flow occurred two weeks before the operation. Pelvic examination showed the uterus to be retroverted, small, and freely movable; the utero-sacral ligaments were very tense and tender. The preoperative diagnosis was retroversion of the uterus and possibly implantation adenoma of the utero-sacral ligaments.

Operation was at the Albany Hospital, November 28, 1921. The uterus was

found to be retroverted and containing several small leiomyomas. The right ovary was lightly adherent to the side of the pelvis and showed a small pigmented elevation (about 1.3 mm. in diameter) on its lateral surface (Fig. 36). The left ovary seemed normal. A small elevated area about 5 mm. in diameter was present on the posterior surface of the uterus above the origin of the right utero-sacral ligament. Pigmented dots were present in this area. A similar condition was present involving both utero-sacral ligaments. Histologically the right ovary contained a typical hematoma of endometrial type about 4 mm. in diameter, which had probably perforated (Fig. 37). Typical adenomas of endometrial type were found superficially invading the posterior wall of the uterus in the area described above, and also the utero-sacral ligaments. The adenoma involving the posterior surface of the uterus and the utero-sacral ligament could have arisen from the ovarian hematoma, or both of these and the ovarian hematoma could have arisen from epi-

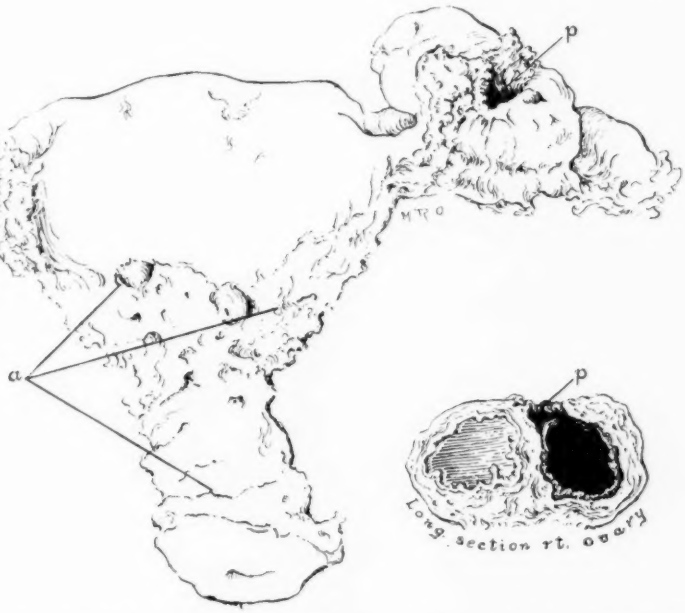


Fig. 61.—(Case 21 of the first series.[1]) Hematoma of endometrial type of the right ovary (Fig. 63), with evidence of a previous perforation, and extensive implantation adenoma of endometrial type involving the posterior surface of the uterus (Fig. 64), and fusing it to the anterior wall of the rectum (uterus retroflexed). Posterior view of the specimen removed at operation (x 3/4); right ovary turned upward exposing the perforation on the under surface, ovary also shown in longitudinal section. On freeing the adherent ovary at operation, the perforation was reopened and some of the chocolate-like contents escaped. The extent of the implantations on the posterior surface of the uterus is indicated by the pointer "a" and is much greater than in the previous illustration, where the perforation was smaller. These implantations could have arisen from epithelium escaping both from the tubes and from the perforation of the ovarian hematoma. I believe that the chief source was from the perforation of the ovarian hematoma (compare with Figs. 59 and 60).

thelium escaping from or through the fimbriated end of the tube. Both tubes were apparently normal and patent. The patient made a satisfactory convalescence.

CASE 12.—*Bilateral hematosalpinx and "adenomyoma" of the distal ends of both tubes with extension through to their peritoneal surface, implantation adenoma of endometrial type on the posterior surface of the uterus, small intramural leiomyomas, of the uterus (no evidence found of hematomas of endometrial type in the ovaries).* Mrs. F. S., aged forty-one, complained of uterine bleeding. She had been married

eleven years and had never been pregnant. Menstruation had been regular and normal until a year ago; since that time it had become more frequent, of longer duration and at times was painful. The last flow occurred three weeks before the operation. For the last six months there had been at times bleeding independent of menstruation. Pelvic examination showed a slightly enlarged uterus with apparent inflammatory masses on both sides. The preoperative diagnosis was a possible intramural or submucous myoma with bilateral hydrosalpinx or adherent tubes and ovaries.

Operation at the Albany Hospital, December 5, 1921. The appendix, both tubes and ovaries and the entire uterus were removed. The ovaries appeared normal, and histologically no trace of adenoma of endometrial type was found in them. The tubes were distended with a thick "chocolate" fluid. The fimbriated ends were occluded, bulbous in appearance, and felt hard (Fig. 5). A diagnosis of

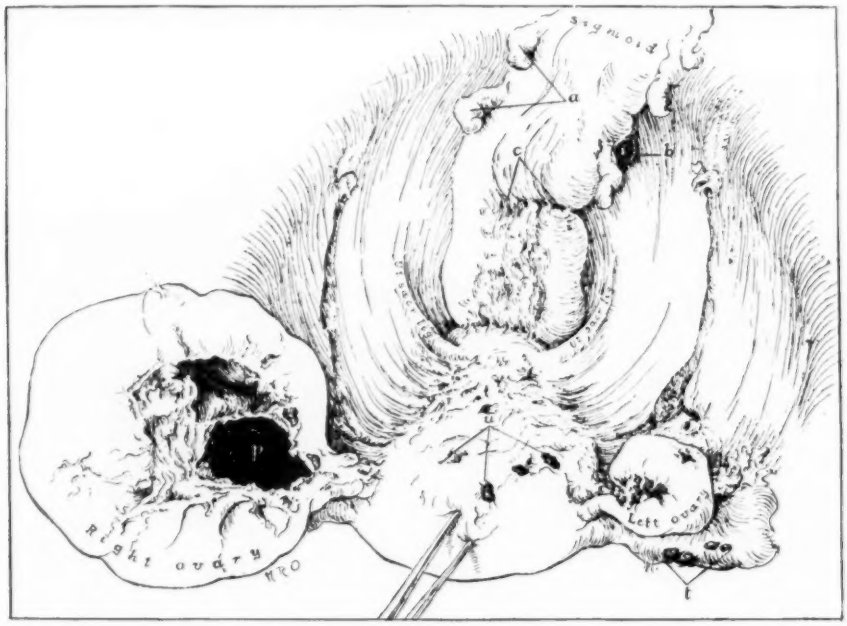


Fig. 62.—(Case 4 of second series[5]). Large hematoma of the right ovary of endometrial type (Fig. 43), with evidence of a previous perforation (*p*) and extensive implantation adenoma involving the sigmoid (*c*), its mesentery (*b*) and epiploic appendages (*a*), the posterior surface of the uterus (*u*), and the left tube (*t*). The condition found at operation is indicated; after freeing the ovarian hematoma, ligating and cutting the ovarian vessels and drawing the uterus upwards and forwards. The distribution of the implantations is similar to that of an ovarian carcinoma with perforation, and I believe that the greater portion or all of the implantations in this case arose from epithelium escaping from the perforation of the ovarian hematoma, just as implantation carcinoma arises from epithelium escaping from the perforation of the primary malignant ovarian cyst. The larger the ovarian hematoma and the larger the perforation, usually the greater the extent of the implantation adenoma associated with it. It is for these reasons that I believe that the ovarian hematoma of endometrial type may be a hot bed, incubator or intermediary host in the origin of implantation adenoma. (Compare with Figs. 59, 60 and 61.)

"adenomyoma" of the distal ends of both tubes was made before they were examined under the microscope. Histologically the distal ends of the tubes resembled a section of an adenomyoma of the tube. Some of the dilated spaces were identical in structure with some of the hematomas of endometrial type found in ovaries. Stromal hemorrhage was present, with loss of the overlying epithelium, and endothelial leucocytes were also present. The histological picture indicated

that a structure similar to an adenomyoma had developed in the distal ends of both tubes, which had reacted to menstruation, causing miniature endometrial hematomas; and some of the blood escaped into the lumen of the tubes, giving rise to a hematosalpinx. The glandular elements extended through to the peritoneal surface of the tubes, and therefore menstrual blood could have escaped into the peritoneal cavity. The blood escaping from the tube might carry with it epithelial cells set free by the underlying stromal hemorrhage rupturing into the lumen of the tube. These epithelial cells might cause implantation adenoma. Such an implantation adenoma, which was similar histologically to the adenoma of the tube, was present on the posterior surface of the uterus where material escaping from the tube would be apt to fall (Figs. 11 and 12). I believe that the adenoma on the posterior surface of the uterus arose from this source. The patient made a satisfactory convalescence.

CASE 13.—*Implantation adenoma (of endometrial type) in the culdesac, adherent right ovary, retroflexion of the uterus.* Mrs. A. McE., aged 30, complained of

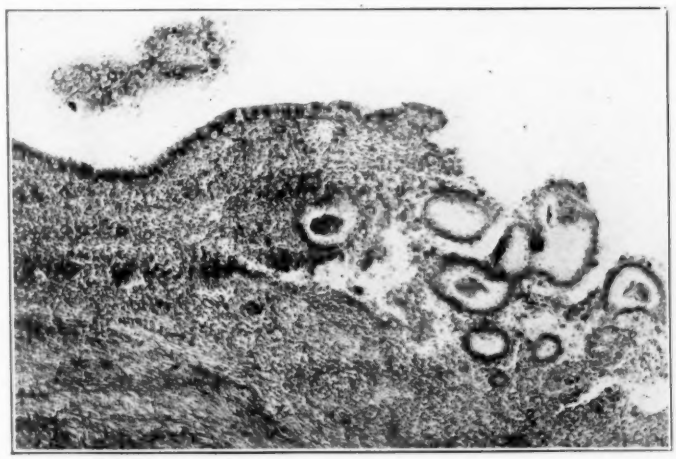

Fig. 63.—(Case 21 of first series[1]). Photomicrograph (obj. 16 mm.) of a section of the wall of the ovarian hematoma near the site of the perforation (Fig. 61). Here the hematoma is lined by columnar cells (ciliated), and gland-like structures are present in the underlying stroma. Histologically it suggests uterine mucosa. The epithelial lining was present only about the site of the perforation and in pockets of the wall of the hematoma, where it had not been completely cast off by hemorrhage (menstruation). The greater portion of the hematoma was lined by a pigmented wall consisting, for the most part, of endothelial leucocytes in various stages of retrogression similar to that shown in Figs. 48 and 49.

sterility and indefinite pains in the lower abdomen. She had been married for eight years, and had never been pregnant. Menstruation was regular, moderate in amount and free from pain. The last menstrual flow occurred two weeks before the operation. Pelvic examination showed the uterus to be retroflexed, possibly adherent. A definite nodule could be palpated in the culdesac to the right of the median line. The preoperative diagnosis was retroflexion of the uterus, and possibly implantation adenoma in the culdesac.

Operation at the Albany Hospital December 17, 1921. The uterus was found to be freely movable; the lateral surface of the right ovary was lightly adherent to the lower part of the posterior layer of the broad ligament. The ovary was freed and carefully examined for any evidence of hematomas, and none was found. Both tubes appeared patent and normal; the left ovary seemed normal. A small nodule about 6 mm. in diameter with puckering of the peritoneum over it, was

found imbedded in the tissues of the culdesac just mesial to the uterine origin of the right utero-sacral ligament. This was excised, the appendix was removed and the uterus was suspended (modified Gilliam operation). Histologically the nodule removed from the culdesac proved to be an adenoma of endometrial type (Figs. 4 and 13). This adenoma may have arisen from the implantation of epithelial cells from the right tube, or from the perforation of a small hematoma of the right ovary (the latter was slightly adherent) which had subsequently disappeared. I believe more likely the former. The patient made a satisfactory convalescence.

CASE 14.—*Pregnancy (early), multiple leiomyomas of the uterus, perforated hematoma (of endometrial type) of the left ovary, implantation adenoma (of endometrial type) of the posterior surface of the uterus; typical decidual reaction of the lining of the ovarian hematoma and of the implantations on the posterior surface of the uterus.* Mrs. A. H. B., aged thirty-seven, complained of indigestion and profuse, painful menstruation. She had been married eleven years and had never been pregnant. Menstruation had always been profuse, but much more so the last three years. At times it had been painful; pain had been more severe and more frequent the last year. The last mentrual period occurred two weeks

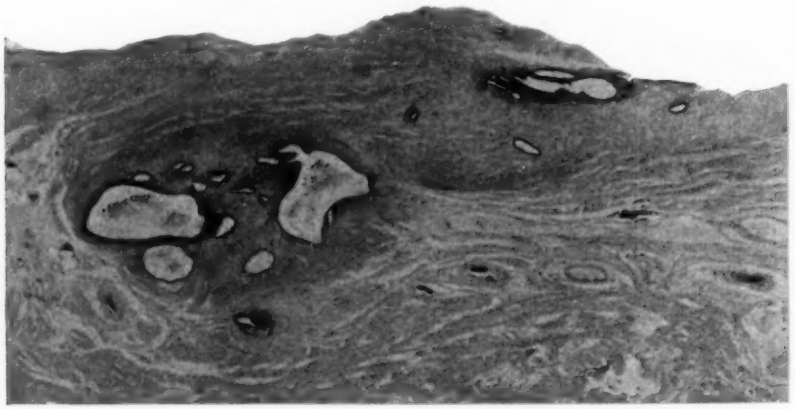

Fig. 64.—(Case 21 of first series[1]). Retouched enlargement (x 15) of a section of the posterior uterine wall shown in Fig. 61. Adenoma of endometrial type is present on the surface of the uterus, invading the wall and giving rise to an "adenomyoma." Some of the epithelium lining the gland-like spaces is ciliated, and is similar to that lining the ovarian hematoma (Fig. 63). It is an "adenomyoma" arising from epithelium implanted on the posterior surface of the uterus, probably escaping from the perforation of the ovarian hematoma. In my experience the most frequent origin of "adenomyoma" of the uterus is from epithelium implanted on its peritoneal surface, and this epithelium is derived either from the perforation of an ovarian hematoma, which acts as an intermediary host (but not as an essential one), or through the tubes.

before the operation. Pelvic examination showed that the cervix was pushed forward by a hard tumor filling the culdesac. The uterus was irregularly enlarged. The preoperative diagnosis was multiple leiomyomas of the uterus. At the operation at the Albany Hospital, December 29, 1921, the uterus was found to contain multiple leiomyomas; the largest, being about 10 cm. in diameter, was wedged in the culdesac. The left ovary was slightly enlarged and firmly adherent to the posterior surface of the broad ligament. On freeing it a small amount of chololate-like fluid escaped. The appendix, both tubes and ovaries, and the entire uterus were removed (Fig. 67). On incising the uterus an early pregnancy was found, the fetus being 14 mm. in length (Fig. 68). The corpus luteum of pregnancy was present in the right ovary. The hematoma of the left ovary was about 2 cm. in diameter, and the perforation had occurred in its under surface. The wall of the hematoma

contained a wavy, almost polypoid lining, which histologically resembled in its decidual reaction the compact layer of the decidua vera of the pregnant uterus (Figs. 69 and 70). The epithelium for the most part was lacking, but was present in the depressions between the polypoid elevations (Fig. 69). Small bleb-like elevations were present over an area about 1x2 cm. on the posterior surface of the uterus, adjacent to the portion of the broad ligament to which the ovarian hematoma was adherent. These elevations varied in size from 1 to 3 mm. and were not pigmented like the usual implantations of endometrial type. Histologically they showed a typical decidual reaction, the larger ones with a compact and spongy layer resembling that of the decidua vera of the uterus (Figs. 71 and 72). In this specimen there were three cavities lined by tissue with the typical decidual reaction of pregnancy, the uterine cavity, the hematoma of the ovary and the implantations on the posterior surface of the uterus. I believe all three cavities were lined by similar tissue.

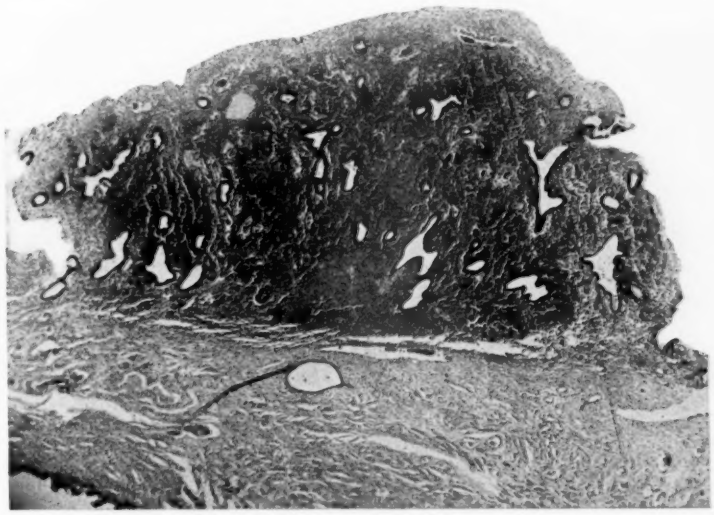

Fig. 65.—(Case 4 of second series[2]). Photomicrograph (obj. 50 mm.) of a section of the implantation adenoma of endometrial type of the left fallopian tube shown in Fig. 62. It resembles uterine mucosa, and probably arose from epithelium escaping from the perforation of the ovarian hematoma shown in Fig. 62.

CASE 15.—*Implantation adenoma of endometrial type on the mesial surface of the right ovary and on the anterior surface of the uterus just below the attachment of the right round ligament.* Miss A. W., aged thirty-six, complained of prolonged and profuse menstruation. This had been of a year's duration. A tumor in the lower abdomen had also been noticed for nearly a year. The last menstrual period occurred three weeks before the operation. Pelvic examination showed a uterine tumor extending upward into the abdominal cavity to the level of the umbilicus. The preoperative diagnosis was leiomyoma of the uterus.

Operation was at the Albany Hospital January 6, 1922. The uterus was enlarged as described above, due to one large leiomyoma (21 cm. in its greatest diameter) and several small ones; the appendix, entire uterus and right tube and ovary were removed. The left tube and ovary appeared normal. An implantation adenoma of endometrial type was present on the anterior surface of the uterus below and mesial to the attachment of the right round ligament (Fig. 9.) A similar adenoma was present on the mesial surface of the right ovary, near the utero-

ovarian ligament. It is unusual to find these ovarian implantations on the mesial surface. The tube in this instance was short, and as the pelvis was filled by the enlarged uterus, epithelium escaping from the tube would be more apt to lodge on the mesial rather than the lateral surface of the ovary. I believe that the implantations on the ovary and on the anterior surface of the uterus probably arose from epithelium escaping from the tube. Had the patient not been operated upon, an ovarian hematoma of endometrial type might have developed, the perforation of which might have given rise to further implantations. The patient made a satisfactory convalescence.

CASE 16.—*Implantation adenoma (of endometrial type) on the lateral surface of the right ovary, the lateral (anterior) surface of the right tube near the fimbriated extremity and on the posterior surface of the uterus; intramural leiomyoma of the uterus.* Mrs. L. H., aged thirty-seven, complained of prolonged and profuse

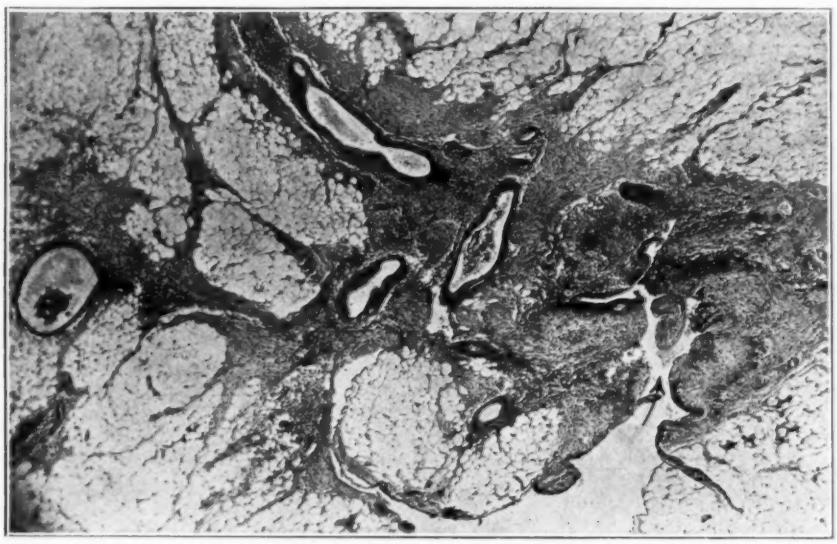

Fig. 66.—(Case 4 of second series[2]). Photomicrograph (obj. 16 mm.) of a section of an epiploic appendage shown in Fig. 62. It demonstrates the invasion of the epiploic appendage by the adenoma of endometrial type, and the great reaction (thickening) of the tissue about the adenomatous tubules. The epithelium lining these tubules was similar to that lining the ovarian hematoma (Fig. 43). Cilia were not found on the epithelium lining the ovarian hematoma, or the implantations.

menstruation. She had been married for five years and had never been pregnant. Menstruation had been regular, moderate in amount, of short duration (2 to 3 days), and free from pain until a year ago. During the last year it had increased in amount and in duration, the more recent menstrual periods lasting from one to two weeks, but being painless. Pelvic examination showed the uterus to be symmetrically enlarged, the fundus extending to the level of the umbilicus. The preoperative diagnosis was leiomyoma of the uterus. At the operation at the Albany Hospital, January 17, 1922, the uterus was found to be symmetrically enlarged and freely movable, except for a small area on the posterior surface (Fig. 7) which was adherent to the sigmoid. This area showed typical adenoma of endometrial type invading the uterine wall. Similar adenoma was also present on the lateral surface of the right ovary, superficially invading it (Fig. 16), and also on the lateral surface of the right tube. The left ovary was normal and both

tubes appeared patent. Both tubes and ovaries, the entire uterus and appendix were removed. Histologically the adenoma invading the posterior wall of the uterus, the right ovary and the lateral wall of the right tube, were similar. The adenomas in this specimen suggested that they had a common origin, namely, from epithelium escaping from the tube. Had the operation not been done a hematoma of the ovary would probably have developed, the perforation of which might have given rise to further implantations. The patient made a satisfactory convalescence.

CASE 17.—*Dilated gland (of endometrial type) with hemorrhage about it on the free surface of the right ovary, implantation adenoma of endometrial type on the surface of the left utero-sacral ligament and the posterior surface of the uterus, which was in contact with the implantation on the utero-sacral ligament (uterus*

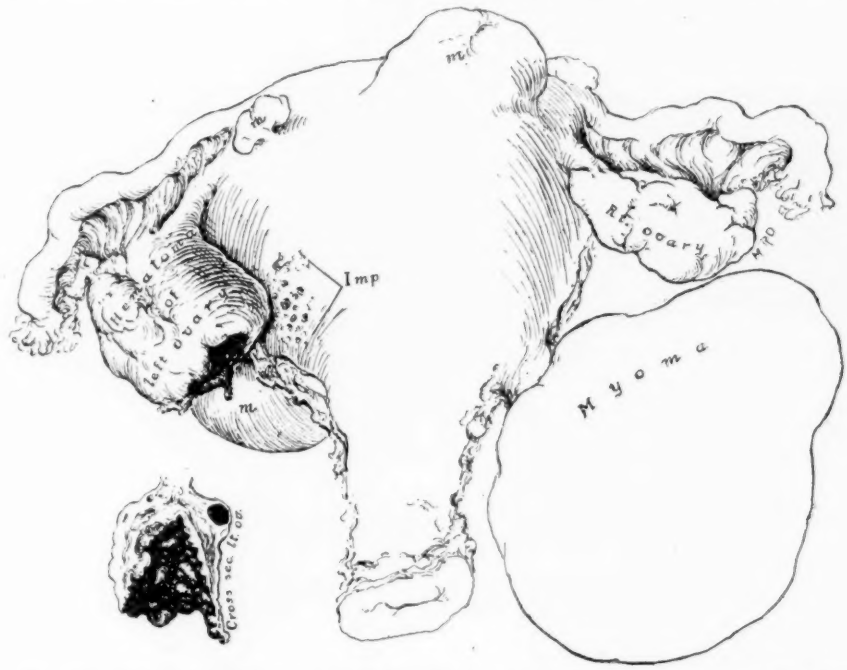


Fig. 67.—(Case 14). Hematoma of endometrial type of the left ovary, with perforation; implantation adenoma (imp.) on the posterior surface of the uterus near the site of the perforation of the ovarian hematoma, multiple leiomyomas, early uterine pregnancy with typical decidual reaction of the lining of the ovarian hematoma, and the implantation adenoma of the posterior uterine wall. Posterior view of the specimen removed at operation (x 3/5).

*in retroflexion), multiple leiomyomas.* Miss M. S., aged thirty-one, complained of severe dysmenorrhoea. Menstruation had always been associated with pain but this had increased in severity the last year. The patient was constipated, but the constipation was not more marked during the menstrual flow. The last flow occurred a week before the operation. Pelvic examination showed a retroflexed uterus which was irregularly enlarged. There was marked tenderness in the culdesac, especially on palpating the utero-sacral ligaments. The preoperative diagnosis was a retroflexed uterus with multiple small leiomyomas, and possibly implantation adenoma in the culdesac. Operation was at the Albany Hospital January 19, 1922. The uterus was retroflexed and contained multiple small leiomyomas. The largest was about 3.5 cm. in its greatest diameter. The appendix, left tube and ovary, and

the entire uterus were removed. A small pigmented elevation about 2 mm. in diameter was noticed on the free border of the right ovary. This was excised with a small amount of ovarian tissue about it. It proved to be a dilated gland of endometrial type with hemorrhage in the stroma about it (Fig. 21). A small corpus luteum hematoma was present in the left ovary, which at the operation was mistaken for an endometrial hematoma. Both tubes were apparently normal and

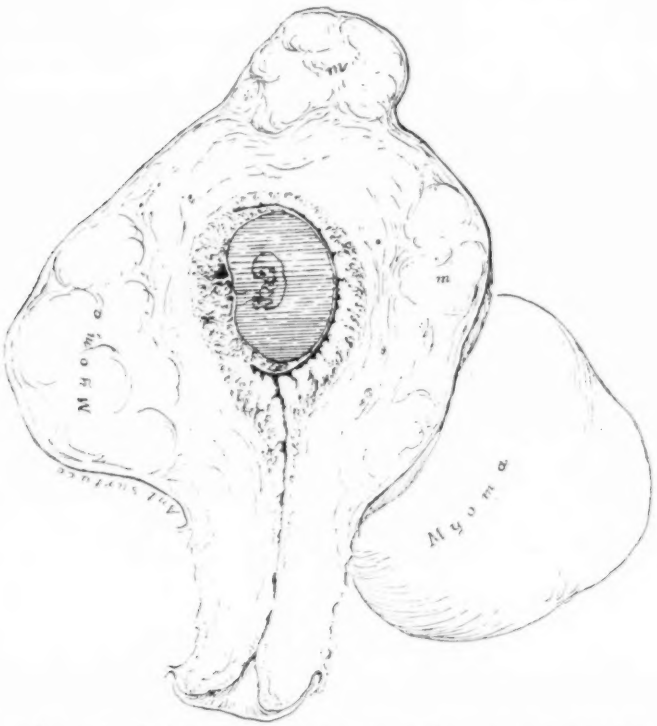

Fig. 68.—(Case 14). Sagittal section of the uterus shown in the preceding illustration, demonstrating the early pregnancy (embryo 14 mm. long) (x 3/5).

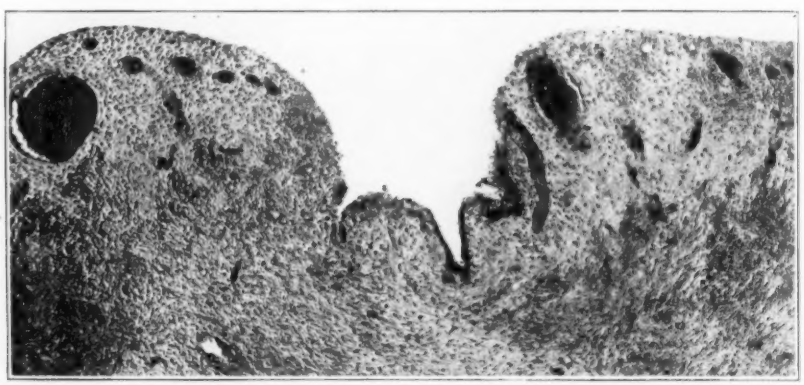

Fig. 69.—(Case 14). Photomicrograph (obj. 16 mm.) of a section of the wall of the ovarian hematoma, showing the wavy, almost polypoid surface, with epithelium only in the depressions. The stroma was very vascular, and showed as typical a decidual reaction as the compact layer of the decidua vera of the pregnant uterus. The surface epithelium, present in the depressions, was also similar to the surface epithelium of the compact layer of the decidua vera.

patent. Implantation adenoma of endometrial type was present on the surface of the left utero-sacral ligament and of the posterior wall of the uterus, which were in contact with each other, the uterus being retroflexed (Figs. 6 and 14). What is the relation between the adenoma of endometrial type on the free surface of the right ovary and those on the surface of the left utero-sacral ligament and the posterior surface of the uterus? They all could have had a common origin from

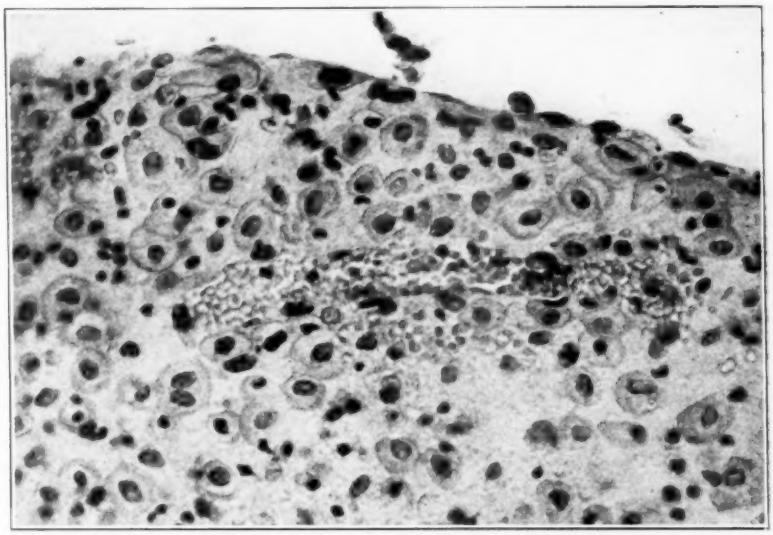

Fig. 70.—(Case 14). Retouched photomicrograph (obj. 4 mm.) of a portion of the stroma shown in the preceding section. Histologically the cells are typical decidual cells.

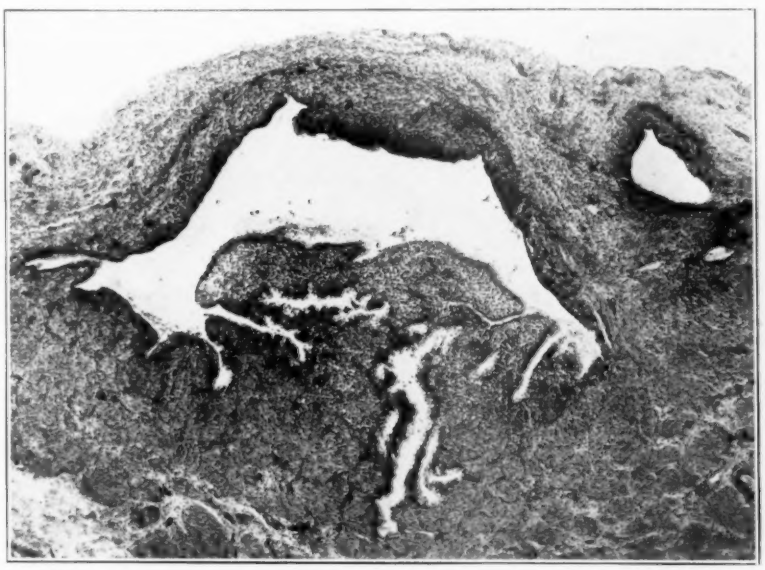

Fig. 71.—(Case 14). Photomicrograph (obj. 50 mm.) of a section through a portion of the implantation adenoma, involving the posterior surface of the uterus (Fig. 67). The larger adenomatous space in the center is a miniature uterine cavity, with a typical decidual reaction of a portion of its lining forming a compact and spongy layer, similar to that found in the decidua vera of the pregnant uterus.

epithelium escaping from the tube and becoming implanted on the surface of the ovary and the pelvic structures. The adenoma on the surface of the right ovary might represent the remains of a small ovarian hematoma in which perforation, with the loss of the greater portion of the epithelial lining, had been followed by repair. The patient made a satisfactory surgical convalescence.

CASE 18.—*Implantation adenoma (of endometrial type) of the posterior uterine wall, culdesac, and posterior surface of the right broad ligament; retroflexion of the uterus.* Mrs. H. D., aged twenty-two, complained of severe dysmenorrhea. She had been married for sixteen months and had not become pregnant. She was very anxious to have children. Menstruation had been regular, moderate in amount and

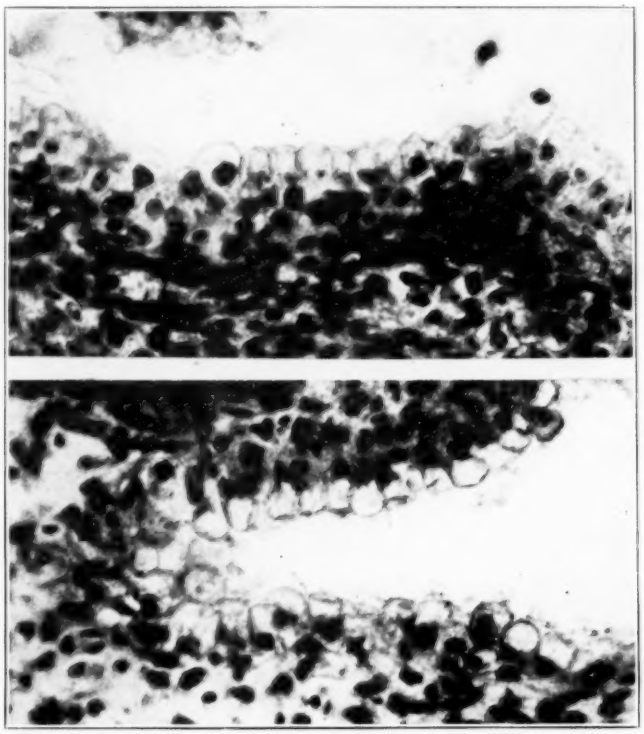

Fig. 72.—(Case 14). Two photomicrographs (obj. 4 mm.) showing the character of the epithelium lining the glands in the spongy layer of the implantation adenoma of the posterior uterine wall, shown in Fig. 71, and that lining the glands of the spongy layer of the decidua vera of the pregnant uterus. Histologically they are identical. The typical decidual reaction of pregnancy was present in the lining of three separate cavities of this specimen: the uterine proper, that of the ovarian hematoma, and that of the implantation adenoma of the posterior uterine wall. The uterine cavity proper was lined by endometrium. By what term should the lining of the other two be designated? Sections taken from three different portions of both fallopian tubes failed to show any decidual reaction.

had been associated with pain ever since puberty. For the last two years the pain had been increasing in severity, and lasted for the entire duration of the flow. The last flow ceased the day before the operation. Pelvic examination showed that the uterus was retroflexed but freely movable. The preoperative diagnosis was retroflexion of the uterus. Operation at the Albany Hospital March 18, 1922. The appendix was first removed. The uterus was found to be retroflexed and after replacing it a pigmented (hemorrhagic) elevation about 2 mm. in diameter was noticed on the posterior surface of the right uterine cornu, and a similar but

broader elevation (implantation) was detected in the culdesac (Fig. 3), at a place which exactly came in contact with the implantation on the uterus when the latter was replaced in retroflexion. A similar implantation (but not as hemorrhagic) was found on the posterior surface of the right broad ligament. Both tubes and ovaries appeared normal, and the latter were examined very carefully for implantations. The implantations described above were excised, the uterus was suspended and the cervix dilated.

Histologically the three implantations were all adenomas of endometrial type. All had apparently reacted to menstruation, the one on the uterus and the one in the culdesac to the last menstrual period, and the one on the posterior surface of the broad ligament to a previous period (Figs. 17, 18, 19 and 24). These implantations were in situations which at times could have been in contact with the fim-

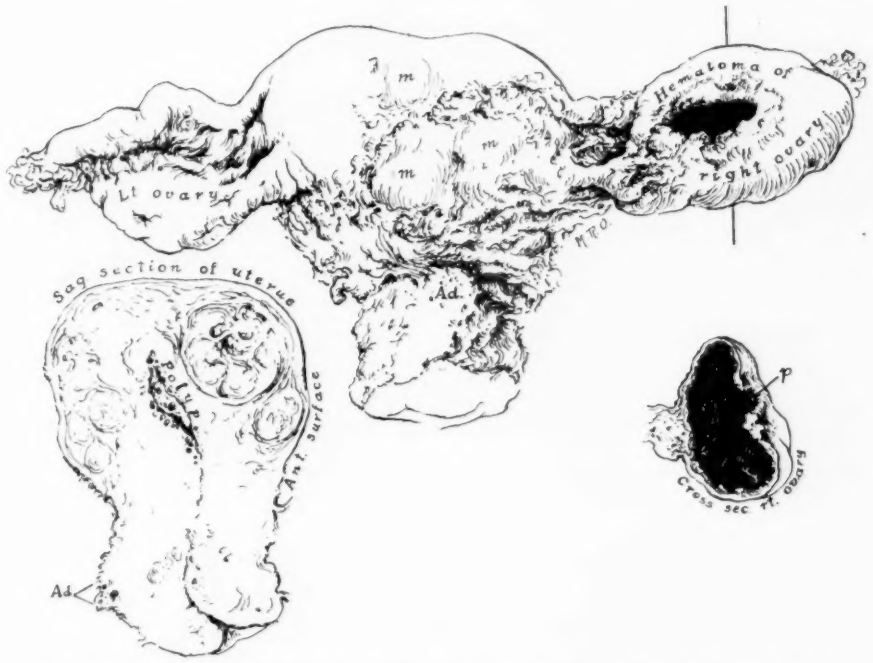


Fig. 73.—(Case 6). Remains of a hematoma of endometrial type (with evidence of a previous perforation) of the right ovary in a patient 61 years old; implantation adenoma involving the posterior uterine wall, multiple leiomyomas, uterine polyp, uterus retroflexed and fused to the bottom of the culdesac. Posterior view of the specimen removed at operation, right ovary turned upwards exposing its lateral surface with the perforation; ovary also shown in cross section and uterus in sagittal section (x 2/3). See Figs. 74 and 75 for photomicrographs of the wall of the ovarian hematoma and the posterior wall of the cervix at *Ad.*

briated end of the right fallopian tube, and I believe arose from epithelium escaping from the tube. The implantation on the surface of the uterus might have been a contact implantation from the one in the culdesac, or vice versa. The patient made a satisfactory convalescence.

CASE 19.—*Implantation adenoma (of endometrial type) of the mesial surface of the right ovary, the right broad ligament between the tube and the ovary and on the suspensory ligament of the ovary; retroflexion of the uterus.* Mrs. J. McG., aged thirty-two, complained of backache and repeated abortions. She had had three abortions in four years, all occurring at the second to the third month of pregnancy, the last one took place six months ago. Menstruation had been regular,

moderate in amount, and always associated with pain, which had increased slightly in severity. The last flow occurred three weeks before the operation. Pelvic examination showed that the uterus was retroflexed; the appendages on the right side were adherent and very tender on palpation. The preoperative diagnosis was retroflexion of the uterus with pelvic adhesions. Operation was at the Albany Hospital April 6, 1922. The appendix was removed. On exposing the

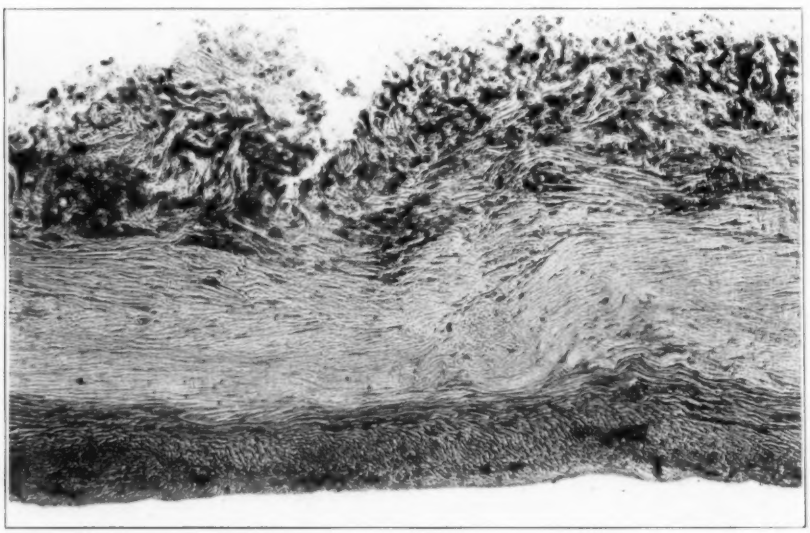

Fig. 74.—(Case 6). Photomicrograph (obj. 16 mm.) of a section of the wall of the ovarian hematoma. The epithelial lining had entirely disappeared. The wall of the hematoma consists of hyaline connective tissue, infiltrated with blood pigment. This represents a later stage of the portion of the wall of the hematoma shown in Fig. 48. As the reaction to menstruation in these hematomas is destructive, and the repair is slow, hindered by the retention of the menstrual blood in the cavity of the hematoma and the attempt to absorb the blood in its walls, the ultimate tendency of the hematoma is one of retrogression.

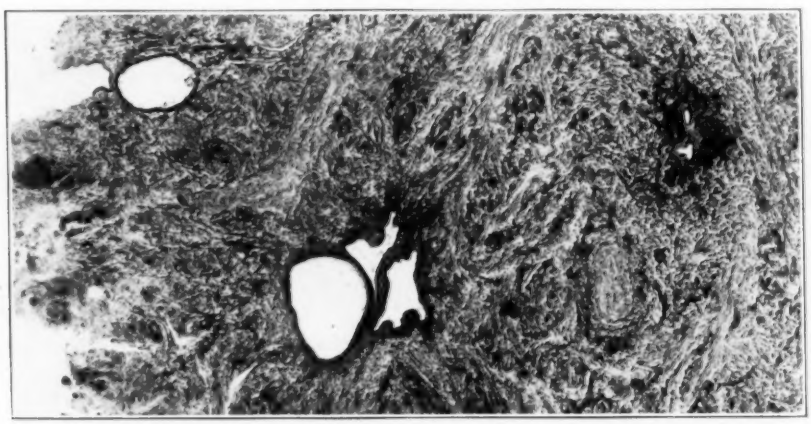

Fig. 75.—(Case 6). Photomicrograph (obj. 16 mm.) of a section of the posterior wall of the supravaginal portion of the cervix indicated by *Ad.* of the sagittal section of the uterus shown in Fig. 73. The adenoma had apparently invaded the cervical wall from its peritoneal surface (to the left), the glandular elements of the adenoma were similar to those of the mucosa of the uterus. The reaction of the lining of the ovarian hematomas of endometrial type and that of the implantation adenomas, to menstruation (except as altered by the attempt to absorb the retained menstrual blood), to pregnancy, and to old age, is similar to that of the endometrium.

contents of the pelvis the uterus was found to be retroflexed, and the right tube was kinked, and adherent to the upper and mesial surface of the ovary in such a manner that the patent fimbriated end rested on the anterior surface of the suspensory ligament of the ovary. Small pigmented elevations of implantation adenoma were present on the surface of the suspensory ligament, directly in front of the fimbriated end of the fallopian tube (Fig. 8). Similar implantations were also present on the mesial surface of the ovary, and on the broad ligament between the ovary and the tube. The left ovary and tube appeared normal. The right tube and ovary, including the portion of the suspensory ligament with the implantations, were removed and the uterus was suspended by suturing the round ligaments to its posterior surface.

Histologically the implantations were of endometrial type. All had apparently reacted to menstruation. The situation of the implantations, especially the one directly in front of the fimbriated end of the tube, on the suspensory ligament of the ovary, would indicate that they arose from epithelium escaping from or through the fimbriated end of the tube. The patient made a satisfactory convalescence.

CASE 20.—*Implantation adenoma (of endometrial type) on the lateral surface of the left ovary and in the culdesac; multiple leiomyomas of the uterus.* Mrs. G. G., aged forty-two, complained of frequency of urination. She had one child eighteen years old, the only pregnancy. Menstruation was regular, moderate in amount, and free from pain until the last menstrual period, which was painful. The patient was operated upon the day before she expected to menstruate. Pelvic examination showed an irregular hard tumor filling the pelvis, and extending upward into the abdomen nearly to the level of the umbilicus. The preoperative diagnosis was multiple leiomyomas of the uterus. Operation was at the Albany Hospital April 15, 1922. The appendix, the left tube and ovary, and the entire uterus were removed. The right tube and ovary appeared normal. A small area with raised pigmented dots was found on the posterior surface of the lower portion of the right broad ligament, near the uterine origin of the utero-sacral ligament; this was excised. The small elevations about 1 and 2 mm. in diameter were present on the lateral surface of the left ovary (Fig. 22). These were bright red in color, apparently due to a recent hemorrhage (the patient expected to menstruate the following day). Histologically these elevations were due to hemorrhage about a gland of endometrial type (Fig. 23). The patient made a satisfactory convalescence.

## CONCLUSIONS

Next to leiomyoma of the uterus, the pathologic conditions arising from the implantation of epithelium which escapes from the fallopian tubes into the peritoneal cavity probably furnish the most frequent pelvic lesions found in women between the ages of thirty and the menopause. During the last year thirty-seven cases with these lesions were found by me in 170 abdominal operations for pelvic conditions in women between thirty and fifty years of age. Should the epithelium escaping from the tube fall on suitable "soil" it develops into glands or tubules of endometrial (müllerian) type which generally react to menstruation. These adenomas are usually

found on the structures which are most frequently in close contact with the fimbriated end of the tubes, such as the lateral and under surfaces of the ovaries and the peritoneal surface of the structures in the culdesac (see text). Implantation adenoma may occur only on the surface of the ovary or ovaries, or both in the ovaries and on the pelvic peritoneum, or on the pelvic peritoneum alone.

The primary peritoneal implantations are usually small and insignificant, but may spread and become invasive.

The implantations on the ovary invade the tissues of that organ, and, as a result of their reaction to menstruation, develop into superficial or deep hematomas (hemorrhagic or menstruating cysts) of endometrial (müllerian) type. The casting off of all of their epithelial lining by menstruation may cause the death of the hemorrhagic cyst before perforation occurs; but most of them rupture, or perforate into the peritoneal cavity. Perforation occurs in the superficial ovarian hematomas while they are still small, a few millimeters in diameter, and as the result of menstruation and perforation the entire epithelial lining may be cast off and the hemorrhagic cyst may disappear.

The hematomas developing in the deeper tissues of the ovary may attain a large size, several centimeters in diameter, before perforation occurs. As the menstrual blood is retained in the cavity of the hemorrhagic cyst and in the stroma of its lining for a long time, many interesting histologic changes occur in the wall of the cyst in the attempt to absorb the menstrual blood, and to reline the denuded surface by epithelium from that which had not been removed by menstruation. The development and activities of the endothelial leucocytes, which act as scavengers, play an important part in the absorption of the menstrual blood and the deposit of the pigment, derived from this blood, in the walls of the hematoma. Perforation permits the contents of the hematomas to escape into the peritoneal cavity, and may temporarily relieve the embarrassment caused by its retention. The perforation is sealed by the ovary or cyst becoming adherent to adjacent structures at the site of its perforation. The hematoma again fills up with blood at its next reaction to menstruation, and repeated perforations may occur. As the reaction to menstruation is destructive, and as the repair and regeneration of the epithelial lining is accomplished under great difficulties (due to the retention of the menstrual blood), the ultimate tendency of the hemorrhagic cyst is one of retrogression.

In its reaction to menstruation, portions of the epithelial lining are cast off into the cavity of the hematoma, and may be found lying free in its hemorrhagic contents. Adenomas of endometrial type may

be found on the surface of the ovary about the perforation, and in the tissue of the structures adherent and adjacent to it, as well as in situations where the material escaping through the perforation would be apt to lodge. This indicates that these adenomas may be derived from the implantation of epithelium cast off by menstruation into the cavity of the hematoma, and escaping through the perforation. Implantations may arise from small as well as from large ovarian hematomas; generally the larger the hematoma and apparently the larger the perforation, the greater the distribution of the implantations from this source. These secondary implantations often resemble normal endometrium more closely than the epithelial lining of the original ovarian hematoma, and are often more invasive, and more closely resemble normal endometrium than the implantations found in the pelvis without evidence of an ovarian hematoma with perforation, i.e., those resulting from a primary implantation from or through the tube. For these reasons I consider the ovary as an incubator, hot bed, or intermediary host in the development of pelvic implantation adenomas of endometrial type, which in some instances may possibly impart greater virulence to the epithelium developing in it; but it is not an essential intermediary host in the origin of all implantation adenomas of endometrial (müllerian) type.

May the primary ovarian and peritoneal implantations (those developing from epithelium escaping from the fallopian tube) arise from both tubal and uterine epithelium? The specimens which I have studied would suggest that they may. We may histologically divide these implantations into three groups. First, those consisting of glands, or tubules and dilated tubules, often lined by ciliated epithelium and without the characteristic stroma of normal endometrium, or with the stroma poorly developed. The structure resembles that of the mucosa of a primary adenomyoma of the tube, and strongly suggests that the implantations might have been derived from the epithelium of the fallopian tube. In the second group the adenomas consist of stroma and glands, similar to those of normal endometrium. The histological picture strongly suggests that these adenomas were derived from uterine epithelium escaping through the lumen of the fallopian tube, namely, from menstruation with a back flow into the peritoneal cavity, or from portions of tubal mucosa which had reacted to menstruation. In the third group the picture suggests a mixture of adenomas of tubal and uterine type, or represents transitional stages from one to the other.

The epithelial lining of the ovarian hematomas or hemorrhagic cysts may also suggest either a tubal or a uterine origin.

In cases with implantation adenomas in the pelvis which are associated with an ovarian hematoma showing evidence of perforation,

both primary implantations from or through the fallopian tubes and secondary implantations from the ovarian hematoma may be present; but the latter probably usually predominates.

It is difficult to determine the factors which favor the implantation and growth of tubal and uterine epithelium on the surface of the ovary, and on the peritoneum. As implantations result from the perforation of the ovarian hematoma, which contains menstrual blood, this may be an important agent in facilitating the development of these implantations. This suggests that menstruation with a back flow through the tubes into the peritoneal cavity may be an important contributory factor. These implantations are frequently found in patients with retroflexion of the uterus, leiomyomas and uterine polyps; conditions which with patent tubes might favor a retrograde menstruation.

The reactions of the lining of ovarian hematomas of endometrial type to menstruation, pregnancy (one case) and old age (two cases) were similar to those of the uterine mucosa.

I believe that the implantation adenomas in the ovary derived from tubal and uterine epithelium are the source of many ovarian cysts and carcinomas, and am convinced that two of the latter, which I am studying at the present time, arose from this source.

I wish to thank those who have made this work possible: Dr. Thomas Ordway and Dr. Victor C. Jacobson, for placing at my disposal the facilities of the pathological laboratory of the Albany Hospital and the Albany Medical College; Miss Miriam Ruth Oliver for her cooperation and skill in presenting the various pathologic lesions as they really appear, and Dr. Lawrence De Noyelles for the preparation of the microscopic sections.

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180 Washington Avenue. (*For discussion, see p. 561*)

# SOME PHASES OF BOVINE GENITAL INFECTIONS OF POSSIBLE INTEREST TO THE MEDICAL PROFESSION*

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THERE are no specific venereal infections which are known to be intertransmissible between cattle and people. It is not impossible that certain genital infections of cattle and men, not designated as specific, may be closely allied or identical.

Aside from such considerations, cattle offer in many respects unequalled opportunity for the study of the physiology and pathology of reproduction. Such a study should prove of great value in determining some of the fundamental principles of sex hygiene and disease.

The internal genital organs of adult cattle are readily and intimately palpable per rectum, surpassing in this respect those of any other animal. The ovaries are readily reached, fully exposed and clearly defined. The development and rupture of the ovisac, the concurrent uterine engorgement and the growth and atrophy of the corpus luteum can be traced as clearly as if the removed organs lay before the student upon the table. Estrum is short, sharp and clear and its relation to ovulation, fertilization and menstruation is too clear to afford room for conflict of opinion.

Genital lesions in great abundance and endless variety can be intimately studied from beginning to end. There are slaughtered annually for food many millions of cattle of all ages and both sexes. When slaughtered they are presumably in good physical health at least insofar as affecting the safety of their carcasses for human food. From these the genitalia are procurable immediately after slaughter by bleeding, without opportunity for the postmortem migration of bacteria. They furnish every imaginable variety of genital disease. The females include nonpregnant animals and those pregnant in every stage.

The sexual relations of cattle are under the arbitrary control of the breeder and offer extensive opportunity for the study at close range of the effects of polygamy, polyandria and sexual excess.

The genital infections of animals bear no burden of social stigma and their results find expression in terms of scientific interest or of

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individual or state economy. While specific venereal infections exist, those not so regarded, but rather analogous to, and not clearly separable from, wound infection are of greater scientific interest because they supply a far richer field for studying the fundamental principles involved in genital diseases. In the medical profession the social stigma of syphilis and gonorrhea has perhaps tended to cause students, reformers and the public to look upon genital diseases as the result of trespasses upon statutory, moral and ecclesiastic laws and has probably befogged to some degree the underlying scientific principles of the problem as a whole.

Recent researches have shown that bovine genital infections of the type not designated as specific venereal diseases are essentially universal, that they advance or retire in obedience to laws reasonably well known and that they may or may not cause serious harm depending upon the state of the balance between the infection and the power of resistance offered by the individual.

It is freely admitted that the copulatory organs of all species harbor an extensive bacterial flora which does not as a rule seriously imperil the sexual or general health. In cattle the infections are not at all confined to the copulatory area but generally involve the cervical canal, uterus, oviducts, ovaries, testicles, epididymes and seminal vesicles.

Infections of the cervix are commonly recognizable clinically not later than the second parturition. The cervical mucosa becomes swollen, reddened and prolapses through the os uteri externum into the vagina. Heifers in first pregnancy not rarely develop an indurative cervicitis rendering parturition difficult or impossible. The cervical mucus is often excessive and wholly abnormal. Cultures from the cervical canal generally produce growths. The uterine cavity is less frequently involved, but in a majority of cases, the non-gravid organ yields positive cultures. The gravid uterus is not far behind the non-gravid in its bacterial content.

The intrauterine infection concentrates about the os uteri internum and at the apices of the cornua in close proximity to the tubal openings. At these foci notable lesions occur. At the apices of the cornua there is generally a very limited area of endometritis with the endometrium edematous and its surface encrusted. The lesions are most extensive in the nongravid horn where the chorion is less vascular and comparatively passive. The chorion lying within this area is necrotic, dry and dark yellow or brown as shown in Fig. 1. The extent of the necrosis is variable but is virtually universal.

The lesions at the os uteri internum are less frequent but more important. There also endometritis is present, the involved area varying greatly in extent and the contiguous chorion may be necrotic

as seen in Fig. 1. Occasionally the cervical canal is devoid of a seal and there protrude into the vagina portions of the chorion which are necrotic and rapidly decomposing as indicated in Fig. 2 but further forward (toward the ovary) the chorion is not detached, and the embryo may yet live, but abortion is inevitable unless gestation is nearing its physiological close. The pathological expulsion of the bovine embryo or fetus (except for surgical or artificial abortion) is, so far as known, always due to cervical endometritis.

Apical or cornual endometritis, though frequently extensive, apparently does not tend to cause the uterus to expel its contents. Not infrequently in cows there is observed an intense diffuse endometri-

Fig. 1.—Bovine fetal sac and embryo at about 100 days. *A*, amniotic sac; *Al*, allantoic sac; *1*, necrotic tip of nongravid horn of chorion; *2*, necrotic area contiguous to the os uteri internum. (From Diseases of the Genital Organs of Domestic Animals, by the author.)

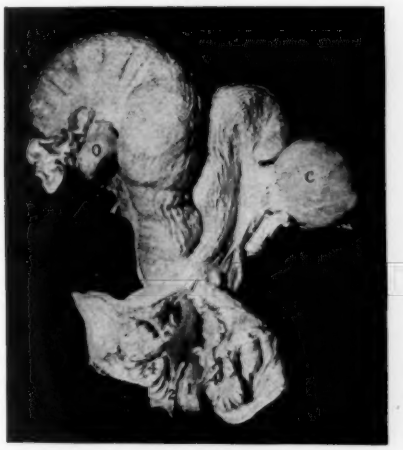



Fig. 2.—Impending abortion in cow pregnant 80 to 90 days. Cervix, uterus and ovaries viewed from above, the cervical canal being laid open to show portions of the chorion occupying the canal and extending into the vagina. *1*, an apparently healthy portion of chorion; *2*, *3*, necrotic chorion; *4*, lips of cervix; *O*, left ovary; *C*, large cyst representing the right ovary. (From Diseases of the Genital Organs of Domestic Animals, by the author.)

tis but instead of causing abortion, it paralyzes the uterus, the fetus dies and undergoes maceration or putrid decomposition.

If the cow is in twin pregnancy the endometritis advancing from the cervix logically involves the basal fetus first. If it dies it will probably not be expelled at once. The presence of the healthy apical fetus inhibits uterine contractions until such period as the infection has killed or seriously injured it, when the two are expelled in quick succession. The basal fetus is smaller than the apical one and perhaps shows maceration. Rarely the two may die in quick succession

and are expelled promptly. Still more rarely the basal fetus is expelled soon after its death and the apical fetus remains to the normal end of pregnancy and is born.

The general conception of the nature of abortion is extremely vague. Abortion is attributed to bad food, mechanical injuries, psychic disturbances, bacteria and other causes, but there is no credible explanation of how so varied a list of factors may each cause the same phenomenon. Such a view leads to confusion in diagnosis, prophylaxis and therapeutics.

My researches indicate clearly that abortion in cows is uniformly attributable to cervical endometritis, which in turn is the result of bacterial invasion. I have examined in the abattoir the uteri of several thousand pregnant cows which had been shipped long distances associated with extreme fright, rough handling and food and water starvation. A number of these showed evidences of impending abortion, always in the form of endometritis radiating from the os uteri internum. I have slaughtered some animals which have aborted following inoculation with the *Bacterium abortus*. In each case an intense cervical endometritis was present. There is no instance recorded in which a cow has been destroyed soon after aborting in which this lesion was not present and in which it did not constitute the sole credible explanation for the disaster. Some veterinarians invoke mechanical injury as a potent cause of those abortions which they cannot attribute to *Bacterium abortus* but rest their assertion upon faith without facts. I had occasion to ship 17 pregnant heifers from Boston, Mass., to Hawaii. Aside from occasionally tumbling over each other in the freight cars the long trip was without incident until transferred at Honolulu to a little interisland freighter which immediately ran into a storm of unusual violence, the vessel being tossed about mercilessly for 32 hours on a sail ordinarily completed in 14. The boat rolled and pitched, the breakers keeping the decks awash and the pregnant heifers were unmercifully thrown about. It was impossible to go among them to feed, water or give other care. They were utterly exhausted when landed but each calved at full term, in a physiological manner and their calves were unusually vigorous and healthy. They did not abort because, and only because, they did not have cervical endometritis. According to my researches no bacterium can directly cause abortion but must first produce an intense endometritis at the cervical end, establishing a peripheral irritation while the ovarian end of the uterus retains its power of effective contraction. This basic lesion having become established, the way is open for the injurious action of a variety of forces. Any psychic, mechanical, bacterial or other factor which lowers the resistance of the pregnant individual may increase the virulence of the

infection upon which the lesion depends and precipitate abortion when otherwise premature birth or birth at full term would have been probable. So among cattle there are observed at times great storms of abortion when in a group of a thousand or more pregnant females more than 90 per cent abort, the immediate cause of which appears to be a bad state of nutrition due to insufficient or unsuitable food but back of all this must be the basic bacterial lesion.

If the principles involved in abortion in cattle as revealed by researches hold true by analogy for woman they tend to clarify the conception of that phenomenon. Probably of greater practical interest this view suggests to the gynecologist that in handling cervicitis in women, the aim should be to eradicate the cervical lesions as completely as possible before fertilization is attempted, not merely to render conception more probable but also to insure the safety of the desired pregnancy.

A highly interesting observation in bovine genital infections is the prevalence of bacteria in the alimentary tract of the fetus which naturally persist at birth. These bacteria are essentially identical with those which exist in the cervix, oviducts, nongravid and gravid uterus. The evidence indicates quite clearly that the infection is swallowed. As soon as the oral end of the alimentary tract is open the embryo begins to swallow its amniotic fluid. The bovine fetus being heavily covered with hair and this being abundantly shed, the results of this swallowing process are strikingly shown in the meconium at full term as seen in Fig. 3 with its luxuriant content of hair. This filtration process usually keeps the amniotic fluid free from bacteria but it appears clear from their presence in the alimentary contents that, like the hairs, they must have been swallowed, suspended in the amniotic liquid. It is most probable that the penetration occurs through the necrotic areas of the fetal sac at the apices of the cornua and that the bacteria reach the allantoic fluid whence there remains only the very thin amniotic membrane to pass in order to reach the amniotic fluid as shown in Fig. 1 and are then swallowed. Once in the alimentary canal the bacteria may acquire pathogenic force, irritate the mucosa and cause fetal diarrhea, or penetrate the mucosa and reach the blood stream. More commonly they fail to acquire pathogenic force and remain inert in what may be termed a state of inclusion rather than infection.

Fig. 3.—Pellet of meconium from new-born calf showing abundance of swallowed hairs. (From An. Rep. N. Y. State Veterinary College at Cornell University.)

A large proportion of aborted fetuses suffer from diarrhea prior to their death and many premature calves are born smeared over with feces. Occasionally a calf born at full term shows that it has had diarrhea as a fetus and the phenomenon may still be active at the time of birth. Some calves are born at full term suffering severely from sepsis and quickly perish.

Most calves are born apparently healthy and vigorous but many of them break down in a few hours to a few days with profuse diarrhea. The probability of the advent of diarrhea depends upon several factors. It is most probable in calves, the dams of which have severe uterine infection as indicated by slow parturition owing to uterine inertia as the result of severe intrauterine infection, and in those dams which showed marked clinical evidences of extensive endometritis and of placentitis as indicated by placental retention. The attack of diarrhea is favored by the early feeding of large quantities of milk, especially if it has been boiled. The phenomenon is best avoided by feeding the raw milk of the mother.

The diarrhea of the newborn is exceedingly interesting because in large herds of dairy cattle the calves are usually removed from their dams either immediately at birth without being allowed to suck or at some period within ten days. The calves are then assembled in large numbers in a calf barn where they are in more or less intimate contact, fed from a common stock of milk by a caretaker without knowledge of, or belief in, the importance of the transmission of bacteria from one individual to another. The entire group in a given establishment is accordingly quite uniformly exposed to any infection carried by any one calf. The result is that in many herds of large size essentially all calves born suffer in varying degrees from diarrhea or from digestive disturbances falling short of this but evidenced by pasty, ill-smelling feces which adhere to the hair and skin of the tail and buttocks. It is not rare to see a mortality of 100 per cent for several months in succession and of 30 per cent extending over a number of years. It is often complicated with pneumonia, pyemic arthritis, pyemic abscesses in the liver, spleen, spinal canal and elsewhere. It is found by experiment that the diarrhea and the associated phenomena may be largely averted by denying the calf all milk or other food for a period of 24 to 48 hours and in the meantime facilitating the emptying of the alimentary tract so that a minimum number of the bacteria included at birth shall remain to multiply in, and decompose the food given. On the same ground, when milk is given, it is in very small amount, not exceeding 2 per cent of the body weight.

Researches have also shown that diarrhea may be largely prevented, or when present, overcome by giving the so-called calf scours

serum, which consists of the blood serum of horses, after the animal has been rendered highly resistant to the introduction of large and virulent doses of the living bacteria presumed to cause the diarrhea. Each progressive step in the researches provides new interest. It is found that while as a rule the raw milk of the mother is the best natural security against diarrhea and that boiled milk tends to cause virulent diarrhea in many calves, those which are born without bacteria in the alimentary tract, or in which the bacteria are very few, as evidenced by an easy birth at full term and a rapid expulsion of the fetal membranes, the calf will grow splendidly upon boiled milk from the first. Or if the alimentary tract of the calf is badly infested with bacteria and boiled milk would probably cause fatal diarrhea, if the horse serum is given liberally the boiled milk may be fed with good results. A further step in the researches shows that when diarrhea is present, it may often be controlled by the hypodermic or intravenous introduction of the blood serum, or the noncoagulated blood or by the transfusion of the blood of the dam.

These data lead to the conclusion that the diarrhea of newborn calves is fundamentally due to bacteria included within the alimentary tract at birth. During pregnancy, the same bacteria may cause fetal diarrhea or sepsis and play an essential rôle in the morbid illness precedent to abortion. In large herds it cannot well be that all calves carry, into postnatal from prenatal life, bacteria which will inevitably cause diarrhea. Observation makes it clear that the disease may have either pre- or postnatal origin.

There has been much discussion in the medical profession regarding the dangers of cow's milk as a food for infants. It has been regarded as definitely inferior for the infant to that of its mother, and the question of whether it should be fed raw, pasteurized or boiled has caused controversy.

The researches upon calves seem to shed much light upon this question. The evidence obtained indicates that while the alimentary tract of the calf commonly carries from the uterus of its dam bacteria in a state of inclusion which possess high pathogenic powers, the fetal blood possesses no adequate protective substances against these bacteria because the placental filter has barred their entrance from the maternal blood. The milk of the mother carries protective substances in a degree which largely meets the demand of the newborn and when the milk is taken as food, the protective substances contained tend to prevent the bacteria in the stomach and intestines from acquiring pathogenic force. But if the milk is boiled the protective substances are destroyed and diarrhea tends to follow. If, however, the calf carries no bacteria in its digestive tract when born, or if such antibodies are experimentally supplied from other sources, boiled milk can be fed with excellent results.

It is to be remembered that the milk of a cow may, and often does, contain the same bacteria as her uterus and hence the same as those in the intestinal tract of the calf. The location of the mammary glands of the cow render it almost unavoidable that portions of any discharges from the genital tract find their way into the milk. They flow from the vulva down along the tail and thighs to the mammae and teats and frequently gain the orifice of the teat canal, pass into the gland and cause mammitis. When the fetal membranes are retained and hang down against the milk glands, infection of the posterior quarters is especially probable. Hence, while the milk of a cow apparently carries valuable protective substances against the bacteria in the digestive canal of her calf at birth, it also is extremely

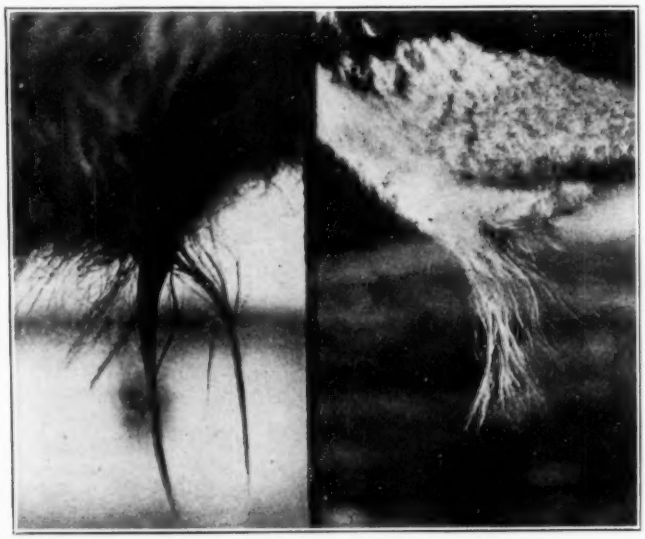


Fig. 4.—Preputial tufts of calves at about 85 days. On the left is shown the matted tuft, stained black, of a dairy calf fed in the ordinary manner on mixed raw milk; on the right is the clean tuft of a calf taken from same herd and fed upon boiled milk. (From An. Rep. N. Y. State Veterinary College at Cornell University.)

liable to contain the same bacteria in more or less dangerous proportions.

That cows' milk regularly contains bacteria which profoundly affect the general health and appearance of calves is quite conclusively shown experimentally. If an apparently healthy bull calf, born in an ordinary dairy herd is fed in the prevailing manner upon raw milk, the tuft of hairs about the sheath opening, at first clean and of the color of its contiguous coat, soon becomes stained black and matted together as shown in Fig. 4. The staining and matting persist throughout life. But the other calf shown in the same figure, taken from the same herd and cared for as nearly as possible in the same manner, except that all milk fed to it had been boiled, shows the tuft of hairs clean, unstained and separate. I have repeatedly tried

this experiment and can find no explanation for the striking difference except that the one was fed on raw, the other on cooked milk. At one time there was so strong a belief current that a calf could not be grown upon boiled milk that the accuracy of my work was doubted.

I then took two calves from their dams at birth and fed them exclusively upon milk held at 30 pounds pressure in an autoclave for one-half hour. The milk was browned to a coffee color by the high temperature. The calves were exceptionally vigorous and grew rapidly as indicated by Fig. 5.

These data regarding calf infections at once raise the question whether they are peculiar to the species or whether they illustrate a common principle and that the genital tract in all its parts and the alimentary tract of the included young are constantly threatened and

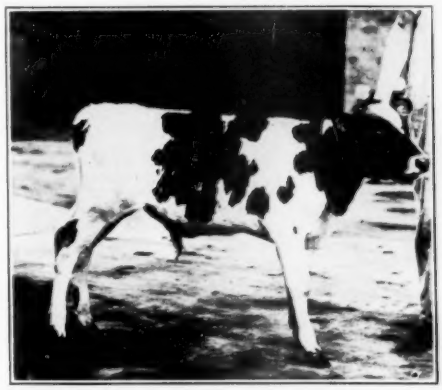


Fig. 5.—Calf at about 85 days grown upon autoclaved milk. The preputial tuft is clean but wet from recent urination.

commonly invaded by bacteria of pathogenic power. So far as researches in domestic animals have gone, the data are in harmony for all. Horses, sheep, goats, swine, dogs and cats suffer like cattle but the studies in these infections have not gone so far and cannot be pursued so readily. Sterility and abortion of adults and diarrhea or other equivalent of the newborn are common alike in all.

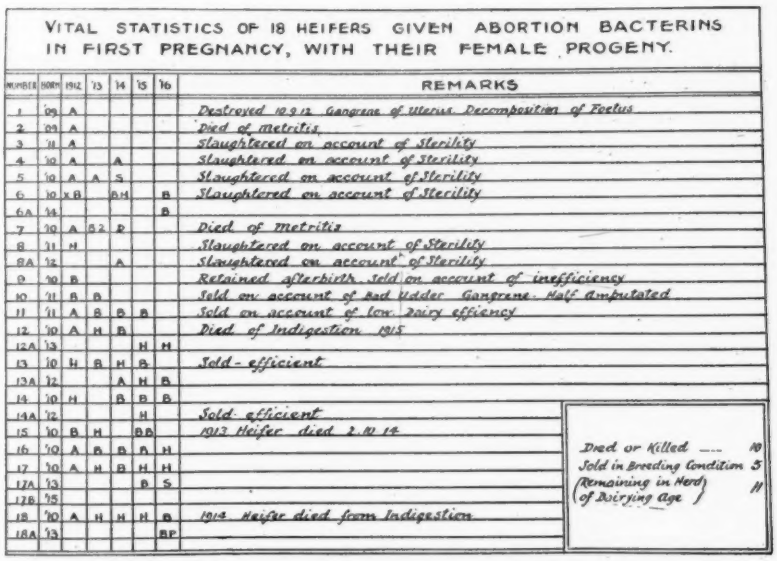
The analogy is not confined to mammals. In the domestic fowl there is a common bacillary invasion of the ovary from which the ova acquire infection. The egg may fail to hatch, but usually incubation is completed with the bacilli persisting in the alimentary tract and yolk sac, where soon after hatching they cause a fatal diarrhea resulting in annual losses which poultry producers compute in millions of dollars.

Members of the medical profession apparently do not believe that genital infections are so common in people. They recognize of course the frequency of syphilis and gonorrhea, which cause an appalling

degree of disease and suffering and great interference with reproduction. In these diseases the uterus and the fetus are presumably invaded by the specific organisms but it seems to be believed that aside from these the uterine cavity and the included fetus and its membranes are commonly sterile. This view is possibly influenced by a belief that the uterus and embryo *should* be germ free. Some may believe that the placental arrangements more effectually bar infection from the gravid uterus and fetus of woman than of the cow. There are considerable variations in placental arrangements in different species of domestic animals but each fails to perfectly exclude infection from the uterus or to guard the embryo. In some respects the placental arrangements of the mare appear more perfect as a bacterial barrier than the placental structures of woman. In the latter the uterine cavity lined by the decidua vera persists until midterm, while in the former the chorion at once comes into relation with the endometrium throughout by means of active placental structures. The only apparent advantage of the placental arrangement in woman is the comparatively inactive layer of decidua reflexa separating the uterine cavity from the chorion, but infection in the uterus and fetus is common and destructive in the former. In each, as in all mammalia, the uterine cavity has three openings which persist throughout pregnancy, the os uteri internum and the two openings of the oviducts. These canals afford the chief bacterial foci in all species from which infection may invade the uterus and embryo. The opportunities for the study of infection in the gravid uterus and the alimentary tract of the fetus are so infinitely superior in cows that this fact possibly explains in part the difference of view. It is not improbable that the view of the medical practitioner that the digestive tract of the newborn child is bacteria-free is based partly upon a study of the meconium near the anus. The study of bovine fetuses in the abattoir shows that culturable bacteria are most numerous in the stomach and fewest at the posterior end of the rectum. Consequently if the medical practitioner bases his conclusion upon a study of the rectal meconium he may fall into error.

The common existence of infection in the gravid uterus of the cow induces the veterinarian to assume the probable presence of infection in the puerperal uterus and to take measures to anticipate its explosion instead of awaiting clinical evidences of disease. Apparently the medical practitioner generally prefers to abstain from invading the puerperal uterus lest he introduce extrinsic infection. Writers upon obstetrics and gynecology make it clear that the medical practitioner is frequently disappointed by unexpectedly finding alarming puerperal infection which he attributes to faulty technic in excluding it, when if the analogy holds true the error may some-

times well be a failure to anticipate injury from an intrauterine infection persisting from pregnancy. The plan outlined for the prophylaxis of puerperal infection in cattle is amply justified by clinical experience, just as clearly, I believe, as is the practice of the obstetrician when he instills silver nitrate solution into the eyes of the newborn to guard against the possible presence of the gonococcus. The analogue of the one-child sterility of the gynecologist is highly destructive in heifers but so far as at present shown may be reduced to a negligible amount by assuming the probable presence of important intrauterine infection in each animal.



VITAL STATISTICS OF 18 HEIFERS GIVEN ABORTION BACTERINS IN FIRST PREGNANCY, WITH THEIR FEMALE PROGENY.

| NUMBER | BORN | 1912 | '13 | '14 | '15 | '16 | REMARKS |
|---|---|---|---|---|---|---|---|
| 1 | '09 | A | | | | | Destroyed 10.9.12 Gangrene of Uterus Decomposition of Foetus |
| 2 | '09 | A | | | | | Died of Metritis |
| 3 | '11 | A | | | | | Slaughtered on account of Sterility |
| 4 | '10 | A | | A | | | Slaughtered on account of Sterility |
| 5 | '10 | A | A | S | | | Slaughtered on account of Sterility |
| 6 | '10 | x B | | B M | | B | Slaughtered on account of Sterility |
| 6A | '14 | | | | | B | |
| 7 | '10 | A | B 2 | B | | | Died of Metritis |
| 8 | '11 | H | | | | | Slaughtered on account of Sterility |
| 8A | '12 | | | A | | | Slaughtered on account of Sterility |
| 9 | '10 | B | | | | | Retained afterbirth. Sold on account of inefficiency |
| 10 | '11 | B | B | | | | Sold on account of bad Udder Gangrene. Half Amputated |
| 11 | '11 | A | B | B | B | | Sold on account of low Dairy efficiency |
| 12 | '10 | A | H | B | | | Died of Indigestion 1915 |
| 12A | '13 | | | | H | H | |
| 13 | '10 | H | B | H | B | | Sold - efficient |
| 13A | '12 | | | A | H | B | |
| 14 | '10 | H | | B | B | B | |
| 14A | '12 | | | | H | | Sold efficient |
| 15 | '10 | B | H | | B B | | 1913 Heifer died 2.10.14 |
| 16 | '10 | A | B | B | B | H | |
| 17 | '10 | A | H | B | H | H | |
| 17A | '13 | | | | B | S | |
| 17B | '15 | | | | | | |
| 18 | '10 | A | H | H | H | B | 1914 Heifer died from Indigestion |
| 18A | '13 | | | | | B P | |

Died or Killed ..... 10
Sold in Breeding Condition 5
(Remaining in Herd) of Dairying Age 11

.—Chart showing Influence of *B. abortus* Bacterins Upon the Prevalence of Abortion.

Fig. 6.—Chart showing low fertility in group of heifers severely ill from diarrhea as calves and carrying severe genital infection until adult age. (From An. Rep. N. Y. State Veterinary College at Cornell University.)

The assumption also that the newborn calf carries pathogenic bacteria in its digestive tract at birth, paves the way for prophylactic measures of great value and makes it possible to generally control the devastating diarrhea and associated phenomena. In doing this, however, precautions against postnatal infections are not abated.

One of the most intensely interesting studies in bovine genital diseases is the influence of prenatal or early postnatal infections upon the fertility of the individual as an adult. Some wideawake breeders promptly destroy prematurely born calves because in their experience they are largely sterile, or if females and they conceive, they probably abort. My clinical observation approves this stand as well as the destruction of calves born at full term but unable to stand up

promptly because of sepsis due to prenatal infection. In many large herds virtually all calves suffer severely from diarrhea and pneumonia and when the surviving females reach breeding age, sterility is common, abortion in first pregnancy is a scourge, retained fetal membranes is very prevalent and unless energetic preventive measures are promptly applied, one-calf sterility is severe. Thus in Fig. 6 a group of 18 heifers representing the entire heifer crop in the herd for the year, all of which suffered severely from diarrhea as calves and were due to calve in 1912 and which ideally should have increased in 1916 to 49 females of breeding age, had decreased to 16 or a deficit from the ideal of 67.3 per cent. In a large herd shown in Fig. 7 where calves suffered severely from diarrhea and pneumonia, out of a group of 593 heifer calves 31 per cent died early and 27.8 per cent eventually became pregnant, of which 44.1 per cent aborted. A change was made in the rearing of calves by which the disease was ameliorated and out of 904 heifer calves, 44.1 per cent conceived as compared with 27.8 per cent and of those in which pregnancy had terminated, only 9.8 per cent aborted instead of 44.1 per cent while

**Calf Scours and Pneumonia, and Abortion and Sterility in Herd B.**

| Time Covered | Heifer Calves Born | | Died of Scours and Pneumonia | Sold as Sterile | Killed account Tuberculosis | Miscellaneous Deaths | Sold for Veal | Conceived | Pregnancy terminated: Calved | Pregnancy terminated: Aborted | In Herd Pregnant | In Herd not Bred |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| May 1 1909 to Aug 31 1912 40 months | 593 | No. | 184 | 8 | 56 | 57 | 118 | 170 | 95 | 75 | 0 | 0 |
| | | % | 31 | 1.4 | 9.4 | 9.6 | 19.9 | 27.8 | 55.9 | 44.1 | 0 | 0 |
| Sept 1 1912 to Oct 31 1916 50 months | 904 | No. | 203 | 6 | 13 | 58 | 0 | 382 | 203 | 22 | 157 | 245 |
| | | % | 22.4 | 0.6 | 1.3 | 6.4 | 0 | 44.2 | 90.2 | 9.8 | 17.4 | 27 |

—The Relationship of Calf Dysentery and Pneumonia to the Prevalence of Abortion in First Pregnancy.

Fig. 7.—Chart showing influence of the health of young calves upon their fertility as adults. (From. An. Rep. N. Y. State Veterinary College at Cornell University.)

17.4 per cent were still pregnant and 27 per cent unbred. A careful study revealed no other reason for the great increase in reproductive efficiency except the improved health of the young calves. These are in entire accord with general observations.

In harmony with this we have found that in experimental calves slaughtered at from 8 to 12 weeks old, those which were vigorous at birth and remained healthy, generally yielded no bacterial growths from cultures made from the cervix, uterus, oviducts or ovaries. On the other hand, cultures were generally obtained from these organs in calves which had suffered from diarrhea or pneumonia, and these cultures corresponded generally with the bacteria commonly found in the gravid uteri of cows. So the conclusion logically followed that diarrhea and pneumonia in calves being due to prenatal, or to early postnatal infection by the same organisms, the bacteria have an affin-

ity for the genital tract. The diarrhea opens a gateway to the blood stream, the bacteria lodge in the genital tract of the calf and lie dormant until puberty. Then in the heifer they invade the ovary from the oviduct through the crater in the ruptured ovisac, cause cystic degeneration of the corpus luteum, which leads to irregular ovulation and consequent irregular menstruation. The intermenstrual pause is irregular and the quantity of menstrual blood variable. With these are associated sterility, abortion, etc. If the analogy holds true even in part, it opens to the medical practitioner a highly interesting and important field of inquiry which has apparently received scant attention. It suggests additional reason for the more careful handling of children, not alone with a view to lowering infant mortality but also to guard the powers of reproduction and through these the general health.

There is a tone of uncertainty in medical literature regarding the part played by the man in sterility and abortion. It is clear of course that destructive disease of the testes or occlusive lesions of their excretory ducts render him sterile. His ability to transmit syphilis and gonorrhea is unquestioned. But outside of these there appears to be no definite understanding. The gynecologist busies himself chiefly with the genital organs of woman and incidentally studies the man but is frequently not clear in his conclusions as to which of the pair is responsible for sterility or abortion. There appears to be a marked general tendency to regard the woman as the chief obstacle instead of regarding husband and wife as equal partners in the problem of procreation.

The polygamy of cattle breeding affords a far better opportunity for observation because the influence of the male can be studied in a large group of females and compared with that of another male in the same herd. Thus in a purebred herd reproduction had been satisfactory for several years when the establishment purchased a young, unused male as an outcross for the heifers sired by the old bull. The young bull came from a herd where the calves suffered much from diarrhea. As soon as he was put into use, trouble, in the form of sterility, abortion, metritis and retained afterbirth, began among his cows while the old herd sire continued to breed satisfactorily. After two years I examined the young bull and discovered a large, indurated abscess of the left seminal vesicle with rectal adhesions, as shown in Fig. 8. The median sagittal section in Fig. 9 shows that the abscess has ruptured into the rectum at 6′. Evidently the bull had been ejaculating bacteria with his semen, but when the abscess formed the contamination of the semen ceased to be possible from all that part of the left gland anterior to the posterior abscess wall.

The result of copulation by this bull is well shown in Fig. 10. In

1918 the original herd sire, designated No. 1, bred satisfactorily, the herd increment being 96.3 per cent. Bull No. 2 then came in. During 1919 and 1920 No. 1 continued to breed well, showing a herd in-

Fig. 8.—Abscessation of left seminal vesicle seen from above. (For explanation of numbers, see legend of Fig. 9.)

crement of 90.0 and 93.3 per cent respectively. During the same period No. 2 showed a high rate of sterility, very high abortion rate (23 per cent) and an abundance of metritis. His herd increment was 46.16 per cent and 57.14 per cent respectively or a trifle over 50 per cent of that of the old sire.

The metritis recorded represents only those cases where it was severe and attracted definite clinical attention.

After bull No. 2 had been killed and it was attempted to breed his cows to No. 1 and to two young bulls, it appears that the infection which the bull had ejaculated in his semen had acquired an enduring

Fig. 9.—Section through abscess of seminal vesicle shown in Fig. 8.
*1*, Pelvic urethra and prostate; *2*, prostatic body; *3*, ampulla of right vas deferens. *3'* cross section of right ampulla; *4, 4*, right seminal vesicle; *5, 5*, left seminal vesicle; *6*, portion of rectum adherent to vesicle; *7*, urinary bladder; *8*, section through left seminal vesicle showing numerous pus areas; *9*, large abscess; *10*, section through sclerotic abscess wall; *11*, fistula extending from the abscess cavity *9*, into the rectum at *6'*.

pathogenic habitat in them. In 3 cows bred to No. 1, not previously bred to No. 2, each conceived with a single coitus while in 15 females which had been bred to No. 2 there was a sterility of 33.3 per cent which with an abortion rate of 6.7 per cent reduced the calvings to 60 per cent.

The picture I have drawn of bovine genital infections, as an endless infection chain might well suggest the possibility of a parent transmitting such contagion to its progeny, and depressing their reproductive powers. Few data are available. In Fig. 11 the breeding records of three sires in one herd are recorded. Bulls Nos. 1 and 2 were of low fertility, averaging one calf for each 3 copulations while No. 3 was of high fertility. The female progeny of No. 1 have averaged 1.8 calves each, No. 2, 1.9 each, and those of No. 3, 3.7 each. The ratio of calves per female for No. 3 equalled the combined reproduction of Nos. 1 and 2. There are many cross currents of infection

**BREEDING RECORDS of HEALTHY and DISEASED BULLS IN A PURE BRED HERD**

| YEAR | BULL | NUMBER COWS | COPULATION | STERILITY | | ABORTION | | CALVES | | METRITIS % (1) | | AVERAGE DURATION OF GESTATION IN DAYS (2) | HERD INCREMENT % (3) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | NUMBER | % | NUMBER | % | NUMBER | % | FATAL | RECOVERED | | |
| 1918 | 1 | 27 | 40 | 1 | 3.7 | 0 | 0 | 26 | 96.3 | 0 | 0 | 285.96 | 96.3 |
| 1919 | 1 | 20 | 22 | 2 | 10.0 | 0 | 0 | 18 | 90.0 | 0 | 5 | 285.06 | 90.0 |
| 1920 | | 15 | 20 | 2 | 13.3 | 1 | 6.7 | 14 | 93.3 | 0 | 0 | 282.79 | 93.3 |
| 1919 | 2 | 13 | 19 | 1 | 7.8 | 3 | 23.1 | 9 | 69.2 | 23.1 | 15.5 | 278.44 | 46.16 |
| 1920 | | 21 | 30 | 4 | 19.0 | 5 | 23.8 | 12 | 57.1 | 0 | 19.0 | 276.0 | 57.14 |
| 1921 | 1(a) | 3 | 3 | 0 | 0 | 0 | 0 | 3(4) | 100.0 | 0 | 0 | | |
| 1921 | 1(b) | 15 | 22 | 5 | 33.3 | 1 | 6.7 | 9(5) | 60.0 | 0 | 0 | | |
| 1921 | 3(b) | 6 | 7 | 6 | 100.0 | | | | | | | | |
| 1921 | 4(b) | 3 | 3 | 2 | 66.7 | 0 | 0 | 1(6) | 33.3 | | | | |

Fig. 10.—Chart showing breeding record of diseased (No. 2) bull of Figs. 8 and 9, compared with the healthy bull (No. 1).
(1) Only marked clinical cases of metritis or retained fetal membranes included; (2) abortions not included; (3) herd increment calves added by birth minus cows dead of genital infections; (4) two had calved and one was pregnant; (5) pregnant at date of report.

in a large herd where polygamy and polyandria are profuse, so that a given stream of infection cannot be accurately traced for a long distance but the limited available data seem to indicate that chronic genital infections are transmitted with a considerable degree of fidelity except scientific measures are taken to control them. In cattle as in men, there are family lines which owing to low fertility tend to become extinct. It is ordinarily attributed to some hazy weakness of reproductive powers. Researches in cattle appear to indicate that in them the explanation lies in the intensity of the genital infection present and its transmission to the progeny.

In the discussion of reproduction and sex hygiene there is frequently heard the term *sexual excess* but so far as I know, no attempt has been made to define it concretely. The polygamy of cattle breeding enables one to observe the direct effects of the frequency of copulation upon the male. In Fig. 11 there is shown a wide variation in the frequency of coitus by the three bulls. No. 1 was evidently highly infected and became useless early. His reproductive efficiency was very low and fell rapidly after he had reached the summit of copulation in his fourth breeding year. No. 2 was looked upon as the most valuable sire and was used far more than the other two combined

CHART OF THREE BULLS IN A LARGE HERD

| NUMBER | YEAR | 1911 | 1912 | 1913 | 1914 | 1915 | 1916 | 1917 | 1918 | 1919 | 1920 | TOTAL | FEMALE PROGENY | CALVES PRODUCED BY FEMALE PROGENY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | COPULATIONS | | 4 | 21 | 48 | 77 | 45 | 16 | 2 | | | 213 | 32 | 41 |
| | % CALVES | | 50 | 48 | 40 | 34 | 22 | 19 | 0 | | | 33 | | |
| 2 | COPULATIONS | 8 | 39 | 82 | 117 | 73 | 106 | 100 | 86 | 64 | 10 | 691 | 85 | 163 |
| | % CALVES | 100 | 33 | 34 | 32 | 27 | 39 | 27 | 26 | 28 | 90 | 32 | | |
| 3 | COPULATIONS | 23 | 70 | 61 | 37 | 35 | 6 | 0 | 3 | 2 | 1 | 213 | 44 | 162 |
| | % CALVES | 83 | 54 | 33 | 41 | 40 | 0 | 0 | 33 | 0 | 0 | 50 | | |

Fig. 11.—Breeding chart of three bulls showing decline of fertility with comparative fertility of their female progeny.

and during several years made more than 100 copulations per annum. They were distributed throughout the year but the breeding was heaviest during the spring. At times certain bulls made 2 and rarely 3 copulations a day. All three bulls (as well as 11 others in the herd) show a somewhat even decline in fertility from the beginning and it appears to be hastened as the number of copulations per annum advances. No. 3, being rather unpopular as a sire, was not so extensively used. His fertility was higher and better maintained, his progeny were more vigorous and far more fertile, and in the second generation he far outranks the other bulls. These fragmentary data appear consistent and so far as can be seen are in accord with general clinical experiences. The frequency of coitus, the fertility, the

security of pregnancy, puerperal infection and fertility of progeny are interdependent.

In studying breeding bulls it is found that the most available data are obtainable from the palpation of the seminal vesicles and the examination of the spermatozoa. Important lesions of the testicles and epididymes are often unrecognizable by palpation. When these become clinically evident the bull is usually hopelessly and completely sterile, but as a rule he is not dangerous, because the lesions inhibit ejaculation and hence the infection remains imprisoned. It is the bull of low fertility that is most frequently dangerous to the female. He is also in peril of becoming hopelessly sterile unless prompt sexual rest is provided.

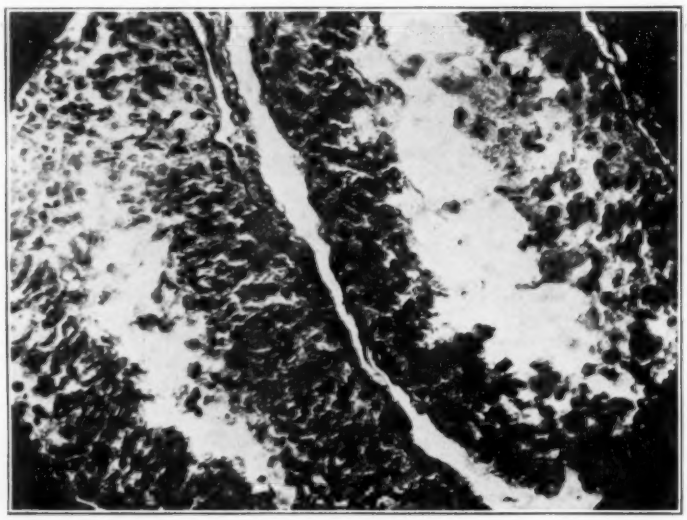


Fig. 12.—Seminiferous tubules showing desquamative orchitis without clinical evidences. In one tubule some spermatozoa are still being formed; in the other the destruction of the germinal epithelium is much advanced.

The two outstanding means for recognizing these dangers are changes in volume, consistency or sensitiveness of the seminal vesicles or abnormalities in the spermatozoa. Sexual activity when it reaches four to seven copulations per week as a rule is soon reflected by the condition of the seminal vesicles but important lesions have already occurred in the mucosa of these before the abnormalities are palpable.

The abnormalities in the spermatozoa may vary widely. Defective motility or total nonmotility indicates high virulence of infection in the seminal vesicles or elsewhere in the course from the testicles to the urethra. Usually the first signal of danger is an increase in the number of defectively formed spermatozoa, such as abnormal form or size of the head. When the abnormally formed spermatozoa reach

about 70 to 90 per thousand, the fertility becomes recognizably low, the pregnancy insecure and the cow is liable to reveal clinical evidences of cervicitis, salpingitis and cystic degeneration of the corpus luteum.

The testicles of such bulls show interesting histological changes. Fig. 12 shows two contiguous seminiferous tubules. In one the germinal epithelium is partly destroyed but spermatozoa are still being formed; in the other the destruction is much farther advanced and its products are chiefly debris and bacteria. In Fig. 13 the destruction is further advanced and the germinal epithelium almost totally destroyed. Such lesions are not clinically recognizable.

Clinically the average bull cannot with safety make nearly 100

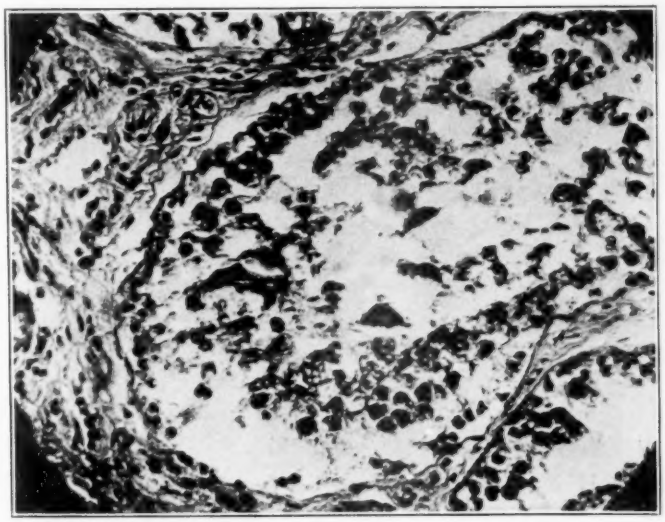


Fig. 13.—Orchitis with almost total destruction of the tubal epithelium.

copulations per annum. Individual sires doubtless may rarely exceed this with safety but this cannot be told except by taking the risk of doing serious injury, both to the prospective fertility of the bull and to the genital health of the cows with which he copulates.

I have not attempted to discuss the identity of the infections mentioned. Most bacteriologists who have studied the problem of reproduction in cattle have limited their researches to one microorganism, the *Bacterium abortus* of Bang and have ignored all other bacteria as of no consequence. The infections here discussed have not, so far as known, any relation to the *Bacterium abortus*. The most prominent organism observed by the very few workers who have paid any attention to the genital flora as a whole, is a streptococcus of the viridans group. There also occur micrococci, staphylococci, bacilli, spirilla, etc. None of them are at present recognized as specific but

rather as belonging to that group of infections which may be designated *general* and not readily distinguishable from the bacteria of wound infection. In a sense they apparently repeat in the genital tract analogous bacteria normally resident upon the skin, virtually harmless upon the unbroken surface with high potentiality for harm when a wound occurs.

The important point is that sexual excess has in cattle a concrete meaning. The data are as yet fragmentary but capable of unlimited extension by research. *(For discussion, see page 563.)*

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# THE TREATMENT OF PUERPERAL SEPSIS BY THE USE OF MERCUROCHROME INTRAVENOUSLY WITH A REPORT OF ANIMAL EXPERIMENTATION IN THE CHEMICAL DISINFECTION OF THE BLOOD*

By Edmund B. Piper, M.D., F.A.C.S., Philadelphia, Pa.†

*(From the Department of Obstetrics and the William Pepper Laboratory of the University of Pennsylvania.)*

THE subject of puerperal sepsis and its treatment has been the cause of acrimonious discussion within the medical profession since the days of Hippocrates. Most of this discussion has been upon the etiology and the predisposing factors in the causation of this condition. Arnold Lea has a very clear cut and a delightful description of the historical side of this controversy. He speaks of "puerperal infection as the general term applied to all infected conditions which arise from the entrance of organisms into wounds of the generative tract in connection with labor or the puerperium," and further states that "all cases of fever during the puerperium, unless clearly attributable to some extraneous cause, should be considered as forms of wound infection." Polak in his monograph on "Pelvic Inflammation" states: "we know from clinical experience that the presence of decidua or a piece of placenta retained in the uterus will not cause endometritis unless infection is also present," and also that "puerperal fever is primarily due to infection of the obstetrical wound by microörganisms."

Mercatus, writing in 1570, was apparently the first observer to mention putridity of the lochia as an essential feature of the disease. In 1843 Oliver Wendell Holmes published his classical essay on the contagious nature of the condition.

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*Read at the Forty-Seventh annual meeting of the American Gynecological Society, Washington, D. C., May 1-3, 1922

†(The author wishes to extend his thanks to Dr. Thomas McMillan of Philadelphia by whom the actual laboratory work was done, both in the original animal experimentation and the clinical controls.)

All students of this condition now agree there must be two things to cause puerperal sepsis. First, the presence of an obstetric wound and second, the presence of a pathogenic organism. Of necessity there must always be the obstetric wound and according to Polak's observation, 50 per cent of patients examined showed bacteria in the uterine cavity six or seven days after delivery. It would therefore appear that if these were the only two factors in the presence or absence of puerperal sepsis, its occurrence should be more frequent than it is at present. Fortunately however, the factor of the individual resistance is probably the greatest determining factor for or against the presence of local puerperal infection.

The term puerperal sepsis is, I believe, used very loosely. From the point of view of treatment alone it would seem necessary to make some distinction between puerperal sepsis, as a blood-stream infection, and as some local infection of the birth-canal. We may say in the old truism we were taught in logic, all cases of puerperal sepsis are puerperal infections but not all cases of puerperal infection are puerperal sepsis.

We are not considering here the absorption of the toxins from retained lochia. We all know that this frequently gives rise to all the symptoms and the graphic temperature chart of a true puerperal sepsis. It would be difficult to differentiate between a puerperal septicemia unproved by positive blood culture and a beginning localization where no localized symptoms have yet presented themselves. We have all seen those cases in which we have, not only a true septicemia but also the local condition as well. The most frequent of these, probably would be streptococcic peritonitis with an infection of the blood stream by the same organism.

As stated before, practically all cases of septicemia are due to infections implanted upon some obstetric injury to the maternal birth canal, but we must not lose sight of the fact that a woman in her early puerperium would be more likely to contract a blood stream infection from some extraneous cause, such as abscess of the teeth, etc., than she would at any other time, though this is rare.

It would seem therefore extremely difficult to differentiate between puerperal infections and puerperal sepsis but as a suggestion it would appeal to me that the latter term should only be used in those conditions, in which there is a definite proof of transmission of infection by the blood stream or lymphatics from the birth canal, and that we speak of every other infection depending upon the child-bearing act as a puerperal infection.

Treatment, in puerperal infection, might be divided into supportive, eliminative, local and blood stream. Under the supportive treatment would come stimulation and feeding. Naturally all cases would

be treated symptomatically. The replies to a questionnaire, sent out by Dr. Hirst, to some of the teaching hospitals, have shown considerable divergence of opinion along different lines of treatment. The question of alcoholic stimulation is fairly evenly divided. We believe in it strongly and use it in large amounts, also the administration of glucose by proctoclysis. We have used nuclein in the attempt to increase the leukocytes, and also autogenous antistreptococcic vaccine, the latter having very excellent results but unfortunately in the very serious cases, which terminate rapidly, we cannot wait for the preparation. In that type of case nothing seems to be of any value.

As to elimination, we keep the bowels open, give water by mouth when possible, otherwise by proctoclysis.

Under local treatment the much mooted subject of the intrauterine douche must be brought up. It is our practice in the University Maternity to irrigate with a return flow intrauterine douche tube those cases which show symptomatically the suspicion of some retention, or where there is a sudden stoppage of lochia, and in those cases where there is an occasionally foul discharge. This is not done with the idea that it is a specific cure for a general infection. We do not believe that we can cure any cases of blood stream infections by this method, but that in this manner we establish good drainage and attempt to keep the endometrium as clean as possible. The force of the flow is not sufficient to do any damage by way of the tubes. We do not believe that we have ever seen any bad results from this practice and we are sure that we have seen many cases rapidly clear up, but it must be understood that it is never done except for a local condition. As to operative treatment, where localization has definitely occurred, I believe that it is generally agreed that, where possible, it should be drained from below, but where there is a definite cornual abscess, suppurative metritis, infection and suppuration of the upper portion of the broad ligaments, tubes and ovaries or localized suppurative peritonitis above Douglas' pouch, we open the abdomen and drain.

Since 1900 there have been many different methods of intravenous treatment for puerperal septicemia, bichloride of mercury, magnesium sulphate, protargol, colloidal silver, eusol, phylacogen, arsenobenzol, formic aldehyde, phenoiodine, ammoniosulphate of copper and others.

Most of these have been reported to be attended with some degree of success. Polak's frequently repeated small blood transfusions have given excellent results in some cases. Antistreptococcic serum has given brilliant results in some cases and others were entire failures When we use it, we do so in doses of 100 c.c. intravenously. Colloidal

silver and arsenobenzol have given us no results whatever but I am informed that in other clinics they have cleared up cases brilliantly.

In the winter of 1920, I had the misfortune to have a patient develop puerperal sepsis. She was a multipara, delivered normally in the hospital without instruments, with, I believe but one vaginal examination before delivery. However, she was admitted to the hospital in labor the same day that a patient died of streptococcic septicemia. The cause of the death in this case was known at the time, and the most careful precautions were taken, and yet this patient was infected and virulently so. The case from the beginning to the end made a peculiarly deep impression upon me because I felt that, had her resistance been better, in spite of the unfortunate fact of the death in the ward, everything would have gone well, but this girl's resistance was below par, both physically and mentally. She had been deserted by her husband some months previously and her mental attitude, when I first saw her was one of entire indifference to her recovery. This factor may be fanciful, but I believe it to be of importance. During the time in which this case was under my observation and care, a great deal of my time was spent in consideration of methods and treatment, and as many before me have no doubt thought, if we could find a germicide which, injected in the blood stream would kill the microörganisms and at the same time not kill the patient, we would be well on the way to the cure of the condition. Young, White and Schwartz in their paper from the Johns Hopkins Urological Clinic on the local use of mercurochrome in infections of the bladder had stated that the toxicity of this preparation had been tested by the injection into the veins of a rabbit. It had been found that in the ratio of 5 mg. per kilo of body weight the animal showed no ill effects, but that in the dosage of 10 mg. per kilo of body weight the animal was killed, and that a dog was able to take 10 mg. without any apparent ill effect. This showed us that we had very little leeway as to dosage.

After a consideration of this report, the following experiments were then carried out by Dr. Thomas McMillan, of Philadelphia, with the aid and valuable suggestions of Dr. Herbert Fox, director of the William Pepper Laboratory. I may say that this work was not entirely unattended by danger. Dr. McMillan was confined to the hospital with a virulent hemolytic streptococcic infection of the hand. His successor in the work, Dr. Hoehn, the pathologic resident at that time, soon after was sent to the Municipal Hospital with scarlet fever. The latter part of the work was done by Dr. McMillan after his recovery.

### GERMICIDAL EFFECTS OF MERCUROCHROME "IN VITRO"

In their original work on mercurochrome, Young, White and Schwartz showed that this drug displayed a marked germicidal action on S. aureus and B. coli when these organisms were suspended in sterile urine.

They found that S. aureus was killed by a 1:5000 dilution of this agent in 5 minutes, while B. coli was killed in 15 minutes.

We attempted similar observations using defibrinated blood as the medium of suspension and a hemolytic streptococcus as the organism. The organism used was from a virulent case of puerperal septicemia. Blood agar slants were prepared from a 24-hour blood culture plate. These were incubated 24 hours.

Using the safe dose for the rabbit, 5 milligrams per kilo of body weight found by these workers, it was thought that in the human body this would represent a proportion of about 1:13,000 of the drug, therefore the dilutions used by us were 1:8000 and 1:16,000. In a 1:8000 dilution of mercurochrome in defibrinated blood, hemolytic streptococci were killed in 40 minutes. In a 1:16,000 dilution, plates prepared at the end of one hour showed two colonies. Those prepared after this showed no growth.

### THE EFFECT OF MERCUROCHROME WHEN INTRODUCED INTO THE BLOOD STREAM OF NORMAL RABBITS

We were guided in the dosage used by the workers referred to above. In accordance with their findings an apparently healthy rabbit weighing 2.4 kilos was given mercurochrome intravenously in the proportion of 5 milligrams per kilo of body weight. The stock solution used was a 1 per cent watery solution. Before the introduction of this drug a urinalysis showed an apparently normal urine. A blood count was done before the introduction of the drug and this gave an entirely normal figure. At intervals after the initial and subsequent doses of this drug blood counts were repeated. Practically no change was noted in the red blood count, and the white blood count was found to vary in the different examinations not more than 3,000 cells. The differential count was not materially changed. Following the initial dose of this drug, identical amounts were given directly into the vein at 24-hour intervals for three days. During this time there was no demonstrable hemolysis as judged by the macroscopic test, hemoglobin determinations, red blood count or urine examinations. After these three injections at 24-hour intervals no drug was given for a day. The injections of the drug were resumed, however, now being given in the proportion of 7 milligrams per kilo of body weight at intervals of 48 hours until three injections

were given. Therefore, in 14 days this animal was given 10.6 c.c. of a 1 per cent solution of mercurochrome intravenously, the last three doses in the proportion of 7 milligrams per kilo. During this time we were unable to demonstrate any urinary changes except a questionable trace of albumin on two occasions; casts were never found. We were unable to demonstrate mercury in the urine. The rabbit was then killed. At autopsy, there were no lesions found in any organ. The kidneys were particularly studied; neither grossly nor microscopically could any pathologic lesions be demonstrated. After an examination of the organs, the skin of the rabbit was removed, the body macerated and examined for mercury. The test was entirely negative.

### THE EFFECT OF MERCUROCHROME ON ARTIFICIALLY INDUCED STREPTOCOCCIC INFECTIONS

A streptococcus recovered from the blood stream of a case of puerperal septicemia was used for these tests. Realizing that the streptococcus lost its virulence when kept for any length of time on artificial media, a blood agar slant was inoculated from a colony of a 24-hour blood culture plate. For the first rabbit used this slant was incubated 48 hours; 2 c.c. of a normal salt solution were then added and the bacterial growth emulsified. One c.c. of this bacterial emulsion was then injected into the ear vein of a rabbit. Blood cultures were taken at intervals from this rabbit. Plates taken 26 hours after inoculation showed the most abundant growth. In this rabbit, blood cultures remained positive until 48 hours after the introduction of the streptococcus; after 56 hours the blood cultures were consistently negative, and the rabbit recovered. Feeling that we had allowed the organisms to remain too long on artificial media, blood agar slants were again prepared from 24-hour blood culture plates of this rabbit and incubated for only 18 hours. Bacterial emulsions were then prepared. Simultaneously 1 c.c. of this bacterial suspension was then injected into the ear of two healthy female rabbits of approximately the same age and size. One rabbit was allowed to run its course untreated as a control and the other rabbit was treated with mercurochrome intravenously.

The untreated rabbit showed positive blood cultures and died within 36 hours. Cultures from this rabbit were then prepared. The heart, lungs and other organs of this rabbit yielded streptococci in great abundance.

The rabbit treated with mercurochrome, twenty-four hours after the introduction of the bacteria, was comatose, lying on its side, with its head markedly everted, and with the cornea of the left eye almost opaque and the sclera much injected. A blood cul-

ture at 24 hours was strongly positive. At this time mercurochrome in the proportion of 5 milligrams per kilo was given intravenously. Blood cultures at 26 hours and 29 hours were strongly positive. Twenty-nine hours after the injection of the bacteria, and five hours after the injection of the mercurochrome, the animal was distinctly better and was sitting erect and eating; the head was still held somewhat everted. Eleven hours after the first injection the head was practically straight and the animal was eating heartily. Twenty-two hours after the first dose of the drug the animal was worse again, lying on its side, breathing shallow and rapidly. The eye condition was very much worse; a panophthalmitis was present with the anterior chamber filled with free pus. Blood cultures taken at this time were positive, 26 hours later or 48 hours after the first injection of mercurochrome, blood cultures were still positive, and mercurochrome in the same strength was administered intravenously. Seven hours after this injection, the blood culture was faintly positive, 24 hours after this second injection the animal was distinctly better, and the blood culture was negative. Blood cultures were taken at frequent intervals after this but no positive cultures were obtained. The cornea of the left eye was very opaque. Feeling that the infection was still active here in spite of the negative blood culture, a third dose of the mercurochrome was given; blood cultures again were negative, and the animal clinically practically well. However, we were misled by the appearance of the eye into believing that pus was still present. It was decided to attempt an enucleation of the affected eye. The animal did not survive the anesthesia. At autopsy we were unable to recover streptococci from any organ, and to our great surprise it was found that although the interior of the eye was destroyed, no streptococci could be obtained. The lungs showed signs of a recent pneumonia but no streptococci could be recovered. We feel that mercurochrome in this instance, saved the life of a rabbit that was comatose with a pneumonia, an eye full of pus, and a blood stream infection, following the introduction into its circulation of a virulent streptococcus,—a strain that caused another rabbit of approximately the same size and weight, to die in 30 hours.

Following the death of the untreated control animal, a bacterial suspension was prepared from a twenty-four hour old blood culture plate of this rabbit.

Another rabbit was then given mercurochrome intravenously in the proportion of 5 milligrams per kilo. One minute after the introduction of this drug into the blood stream, 1.5 c.c. of the streptococcic emulsion was introduced into the circulation. Blood cultures were taken at frequent intervals. These were consistently negative. Ninety-six hours after this procedure, an additional 1.5 c.c. of the

streptococcic emulsion was given the same rabbit in order to see if the effects of mercurochrome persisted. Blood cultures were positive at twenty-two and forty-eight hours. Blood cultures were negative at ninety-six hours.

During the observation on the rabbit treated with mercurochrome, blood cultures becoming negative, our streptococcus was allowed to remain on artificial media too long. Therefore when the next rabbit was injected the culture proved entirely innocuous. In an attempt to increase the virulence of the strain this organism was passed successively through six mice intraperitoneally. The virulence of the organism for mice was greatly increased by this procedure. However, we were unable to gain back a permanent and persistent virulence for rabbits in this organism. Some eighteen rabbits in all were injected with various cultures of this organism but without its ever gaining a uniform virulence. All told, we used over thirty animals, but the results in all cases were not at all conclusive, and, owing to the difficulty of getting uniformity in the animals themselves, other results were not satisfactory.

To sum up, we felt that the experimental work showed that mercurochrome could be introduced into the blood stream of rabbits in certain strengths without any demonstrable harmful effects. We demonstrated also that mercurochrome in dilutions, considerably weaker than those we are able to introduce into the blood stream had distinct germicidal power. We were unable to consistently set up an active blood stream infection in all rabbits. However, we believe that in the case of one rabbit, at least, an animal that was comatose and with an eye full of pus,—this drug manifested spectacular power. Further we believe that in that particular rabbit the drug not only cleared up the blood stream infection but it actually attacked and showed germicidal power upon the local lesions.

Following our experimental work, we felt that we were justified in attempting the use of mercurochrome clinically. But in order to be on the safe side, we at first only tried it on cases that were actually moribund. The first case in which it was used was a woman admitted *in extremis*. Her temperature was 105° F., pulse approximately 160, respiration 40 to 50, she was unconscious and had all the signs of diffuse peritonitis. The drug in her case reacted, as in our later work, with chill, the dropping of her temperature and pulse for a short time and then she promptly died. We tried this again on one or two other cases of a similar nature with the same results. Although these patients died we could see no way in which the mercurochrome had hastened their end, in fact we thought we saw some slight improvement even in these desperately ill individuals.

We used this treatment on other cases, five of which are herewith submitted.

CASE 1.—H. B. Patient admitted to the hospital with a severe thrombophlebitis of the lower extremity. We were unable to obtain a positive blood culture. For days and weeks she ran a typical septic temperature. She was given antistreptococcic serum with no benefit whatever. She gradually became worse and worse until it was thought she could not possibly live more than 48 hours. It was then determined to try mercurochrome intravenously. At this time her leg was almost gangrenous. She was given repeated doses of mercurochrome, of a smaller dosage than we now know can be given safely. The reactions were very marked and she was always clinically better after each dose. At the time that the first dose was given, a blood culture was taken and subsequently found to be strongly positive. Later it became negative under treatment. The patient eventually died of what appeared to be streptococcic pneumonia. At the time of her death, the local lesion in her leg had practically cleared.

CASE 2.—H. S. Patient with a positive blood culture of streptococcus viridans. She was given repeated doses of mercurochrome with rather strong reactions, the

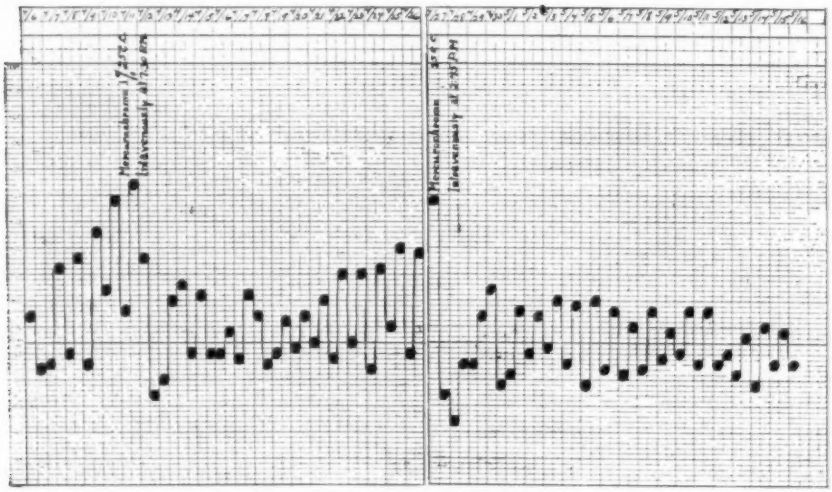

Fig. 1.—Temperature chart from a case of puerperal sepsis, showing high and low daily temperature and recessions after administration of the mercurochrome.

patient always became clinically better. Inasmuch as the results in this type of infection have been uniformly bad, with all kinds of treatment, I did not anticipate any good results. After some weeks of treatment, she insisted upon going home and subsequently it was reported that she died.

CASE 3.—S. S. Puerperal septicemia with a hemolytic streptococcus in the blood. She was treated with mercurochrome with very slight reaction, subsequently she was treated with antistreptococcic serum with no apparent benefit. Eventually the condition localized as a cellulitic phlegmasia of the lower extremity which was opened and drained and the patient made a good recovery. Whether the mercurochrome in this case was of any value cannot be determined but the patient eventually showed a negative blood culture.

CASE 4.—Case of puerperal sepsis of long standing, admitted to the hospital after two months. She had had a thrombophlebitis of the lower extremity which appeared to have improved. Soon after admission she became suddenly worse, the temperature mounting rapidly. Her blood culture showed a hemolytic streptococcus

and she was given mercurochrome. This patient received the largest dose we had ever given up to that time. Her reaction and clinical improvement, as can be seen by the chart (Fig. 1) was very satisfactory. For 12 days her highest temperature at any time was 100.4° F. One dose of the drug was all that was given for a period of two weeks. One week after the first dose her blood culture was negative. Since that time her temperature has risen again, and she was given a second dose, April 27, to which she reacted in the usual manner. Naturally, I am unable to give the end result but she appears to be better.*

CASE 5.—Patient with a moderately severe case of pyelitis. She was not pregnant. She received two doses of mercurochrome three days apart. Her symptoms all disappeared but she still had pus in her urine. Her phthalein elimination was 30 per cent before her first dose and 35 per cent two days after her second dose.

In all cases there are a few points to be noted. The urine in all was typical of any severe infection. The mercurochrome did not change this in any way. The blood picture was in no way changed except that the leukocytes were somewhat increased in some instances. The phthalein test in one case was most satisfactory.

These cases are certainly not definitely conclusive that in mercurochrome we have found a specific cure for blood stream infections, but we feel that we may perhaps after more extensive observation find a way to increase the dosage, so that our subsequent results will be better. In a condition of this kind, in which there is some focus of infection, if we can kill the bacteria that are in the blood stream, the best we can hope for is that by so doing we can give the human organism a chance to cure itself.

The laboratory findings show that the dosage of the drug will of necessity be an important factor. Our original plan gave us a fairly good margin of safety. We gave that dose which was definitely shown to be nontoxic to the rabbit, that is, 5 milligrams per kilo of body weight. That would be about 25 c.c. of a 1 per cent solution of mercurochrome in distilled water for every 125 pounds of body weight. We definitely did not get as good results as we had hoped from this dosage. In case No. 4 as shown above, the dosage was somewhat higher, and based on her case, we feel safe that a nontoxic dose would be 25 c.c. of a 1 per cent solution per 100 pounds of body weight; in her case it was slightly larger. Naturally the body weight is an approximated figure.

The reaction that occurs is almost routinely the same. Within the first hour there may be vomiting; in less than two hours a definite diarrhea will commence. In the first six hours there will probably be a marked chill with a rise in temperature up to 105° F. Following this chill there will be a gradual decrease in temperature with a proportionate pulse rate decrease until the temperature reaches subnormal, gradually rising to normal or slightly above where it should

*Subsequently, this case made a complete recovery and was discharged.

stay from 24 to 48 hours. The diarrhea will continue over a day or so and may require treatment.

Our technic is as follows: A solution of mercurochrome is prepared with sterile distilled water in 30 to 50 c.c. ampoules of a 1 per cent strength. It has been shown by other investigators, which our experience corroborates, that the drug loses its strength unless freshly prepared and for that reason we have it put up in ampoules, after we have determined upon its use. We never carry any solution on hand. It should never be used unless the solution is absolutely clear. The administration is done with a fine needle and a large syringe at a temperature as near 100° F. as possible and the injection made very slowly. Formerly at the end of twelve hours, the patient received a dose of magnesium sulphate. We no longer do this. Her reactions are treated symptomatically. We use external application of heat for the chill and a bismuth mixture as an intestinal sedative for the diarrhea, if it continues too long. A careful record of urinary output must be kept, as it will run below normal owing to fluid output by the bowel. Subsequent administration of the drug may be done. We make it a rule never to repeat as long as there is any sign of the dye in the urine or bowel movements. We have given as many as five doses. Salivation occasionally occurs.

For six months, we had extremely poor results in the few cases in which the drug was used. During that time we gave a dose of salts almost immediately after the injection of the drug. This method was employed in Case 3. It would appear that in so doing we had eliminated the drug before it had a chance and failed to get its germicidal effect.

The reaction that follows the intravenous injection of mercurochrome is analogous to that observed after the introduction of serum, bacteria or sterile milk by a similar route, and is probably explained in the same way. Some of the action of this drug may result from the immobilization or inhibition of the circulating germs, possibly also their direct destruction as in the test tube, aided by an increase of leucocyte activity which always accompanies postinjectional reactions. We have also reason to believe that mercurochrome may reduce the number of circulating organisms by combating them at the localization of the infection. The greatest value of this chemical is known to be in local infection.

## CONCLUSIONS

1. There are certain cases of puerperal septicemia so fulminating and virulent that no treatment can possibly save them.

2. The use of antistreptococcic serum has appeared to be of great value in some cases. Frequently repeated small blood transfusions

are of value, as apparently are other methods of intravenous medication.

3. A solution of mercurochrome given intravenously in the proper dosage, appears, in some cases, to be of great value and to have no deleterious effects.

4. All cases of puerperal septicemia are so serious and so frequently fatal that heroic measures are justified.

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*(For discussion, see page 565.)*

# THE USE OF SUTURES AS TRACTORS IN THE VAGINAL OPERATION FOR PROLAPSUS*

By Thomas S. Cullen, Baltimore, Md.

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THE removal of the uterus for prolapsus is becoming a less and less frequent procedure. The value of the uterus as the keystone in the vaginal vault is more and more appreciated, and, unless malignancy is suspected or there be coexistent pelvic lesions indicating a hysterectomy, the organ is preserved.

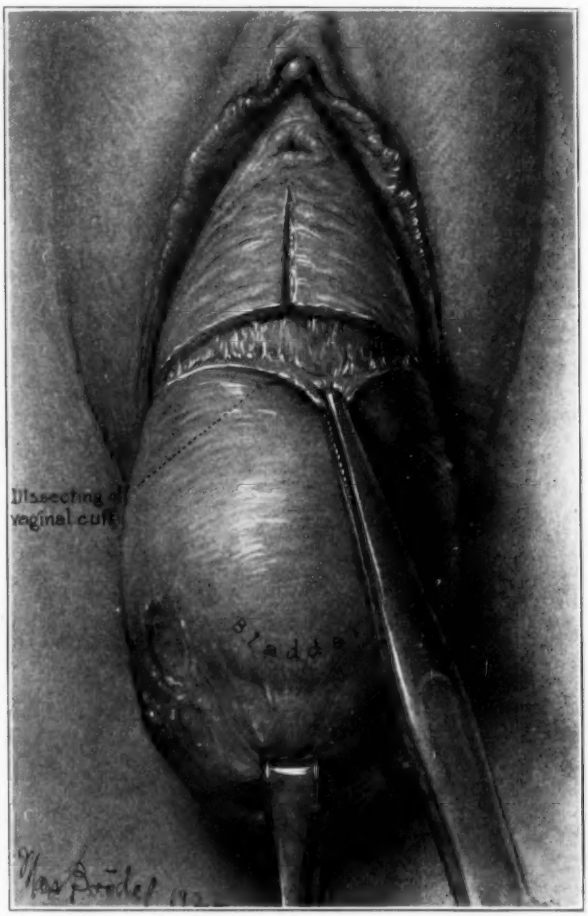


Fig. 1.—Strong traction is being made on the cervix. An incision completely encircles the vagina high up and a perpendicular incision extends from a point just below the urethra to the circular incision. The anterior vaginal mucosa is being dissected down. The bladder extends down almost to the external os.

*Read at the Forty-seventh Annual Meeting of the American Gynecological Society, Washington, D. C., May 1-3, 1922.

The evolution in the improvement in the technic of vaginal operations for prolapsus has been a gradual one and numerous surgeons have had a share in its development. Inseparably linked with this improvement are the names of two distinguished members of this Association, Drs. Thomas J. Watkins and J. Riddle Goffe.

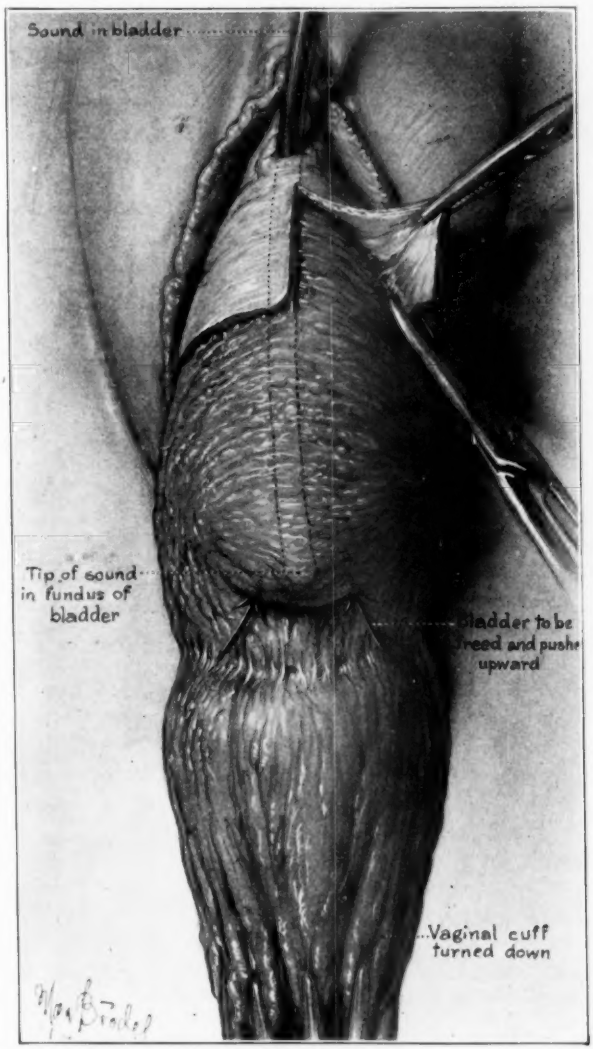


Fig. 2.—The anterior vaginal flap has been dissected down. As a rule the vaginal mucosa of the posterior wall is not disturbed until the cervix has been cut across. The left half of the triangular flap is being cut away.
The sound shows the lower limit of the bladder.

In the few moments at my disposal I shall describe briefly the method which in the main we have found most satisfactory in the handling of uterine prolapsus. It is not new; it has been adapted almost in its entirety from a combination of the work of others. But I shall

also mention a new point in the technic which tends to render the operation a little easier and which at the same time diminishes the bleeding to some extent.

Although to the operator the various stages in the operation may be perfectly clear, to the student or to the onlooker, who is unfamiliar with the procedure it is a difficult puzzle. In the accompanying draw-

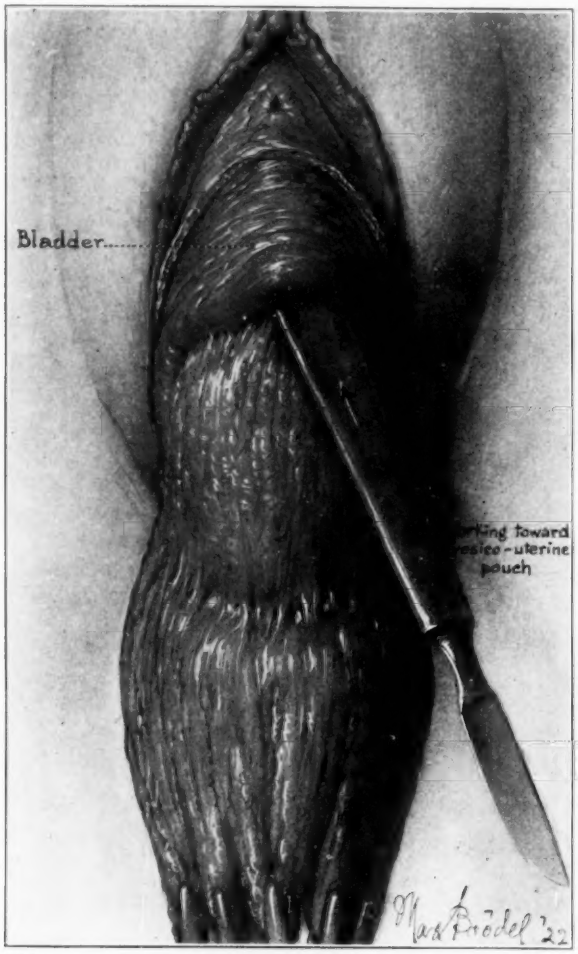


Fig. 3.—The bladder is being carefully dissected free from the uterus. The knife handle is near the vesical reflection of the peritoneum.

ings, made by Max Brödel, every step is rendered perfectly clear, so clear that the student of the future will have little or no trouble in following the operation from beginning to end.

In Fig. 1 we see the markedly prolapsed uterus with ulceration along one side. An incision has been made all the way round the vagina, after which a perpendicular incision is begun just below the urethral orifice and continued down to the circular incision.

In Fig. 2 the vaginal mucosa of the anterior wall has been dissected down nearly to the external os and the triangular vaginal flap on the left side is being cut away. The extent to which the bladder projects downward is indicated.

In Fig. 3 the bladder has been pushed up and separated from the upper part of the cervix and lower part of the body of the uterus.

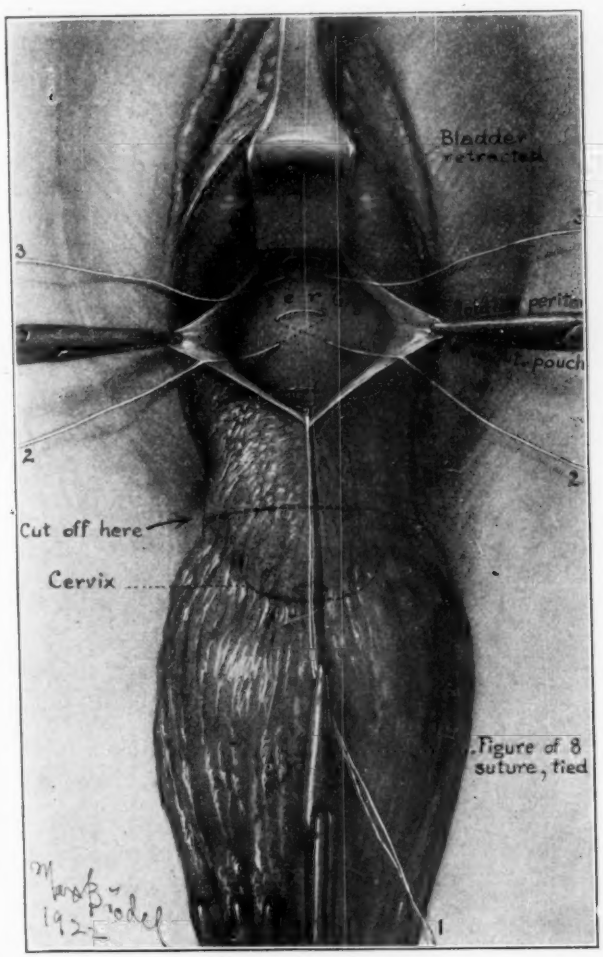


Fig. 4.—Insertion of the figure-of-eight traction sutures which later become the permanent fixation sutures.

Suture *1* has been introduced and tied. Traction has been made on this, more of the fundus exposed and figure-of-eight suture *2* introduced. This is usually tied; traction is made on it, and the third figure-of-eight suture placed and tied.

Any bleeding that may come from the needle holes is usually promptly checked when the suture is tied.

After the peritoneal reflection has been exposed I open it, and then instead of grasping the uterus with a tenaculum or other forceps I place a figure-of-eight suture of chromic catgut in the anterior wall of the uterus, going rather deeply into the wall but taking care not to

enter the uterine cavity. This suture is now tied and used as a tractor. This exposes more of the fundus, and a second figure-of-eight chromic catgut is placed and tied. The first suture is now dropped and traction is made on the second suture. More of the fundus is exposed and a third figure-of-eight is inserted and tied. The entire procedure is shown in Fig. 4.

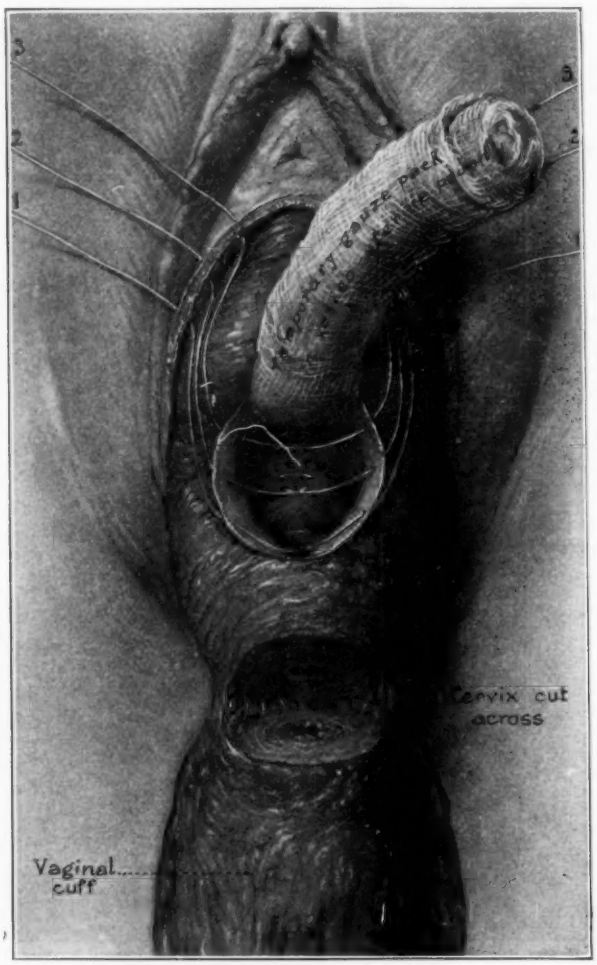


Fig. 5.—A piece of gauze is temporarily placed in the peritoneal opening to prevent the escape of blood from the vagina into the abdomen.

The ends of the figure-of-eight sutures are re-threaded, passed through the peritoneum and then through the edge of the vaginal mucosa, as indicated.

The cervix is now partially cut across, the cervical canal dilated; then the cervix is severed and the vaginal mucosa of the posterior wall dissected away with the cervix.

After the three figure-of-eight sutures have been placed, the end of each is rethreaded, passed through the peritoneum and then through the edge of the vaginal mucosa. This is clearly seen in Fig. 5. After these sutures have been placed, they are clamped, and a small gauze

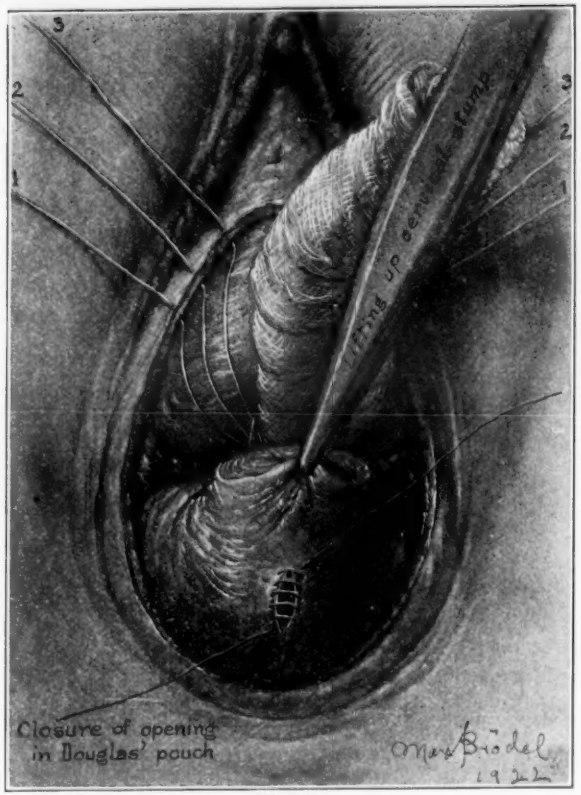


Fig. 6.

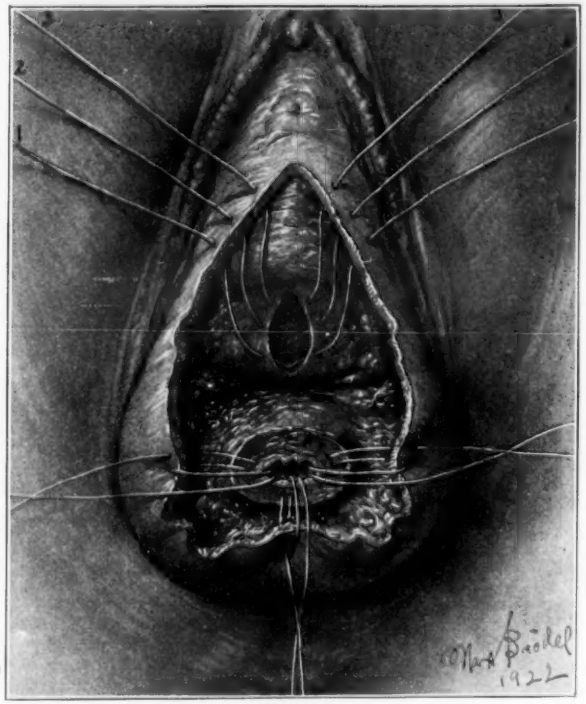


Fig. 7.

Fig. 6.—Closing a small opening into Douglas's culdesac accidentally made when the vaginal mucosa was being dissected free. In some of these cases there is little or no intervening tissue between the vaginal mucosa and the pelvic peritoneum.

Fig. 7.—Approximating the vaginal mucosa to the cervix. Where possible figure-of-eight sutures are employed.

pack is temporarily placed between the bladder and fundus, in order that no blood may escape from the vagina into the peritoneal cavity.

Strong traction is now made on the uterus, and the cervix is cut half way through as indicated. Before it is completely severed, however, the cervical canal is well dilated.

After the cervix has been completely cut through, the mucosa of the posterior vaginal wall, outlined in the primary incision, is dissected free and removed with the cervix.

As indicated in Fig. 6, Douglas's culdesac is occasionally accidentally opened while the mucosa of the posterior vaginal wall is being

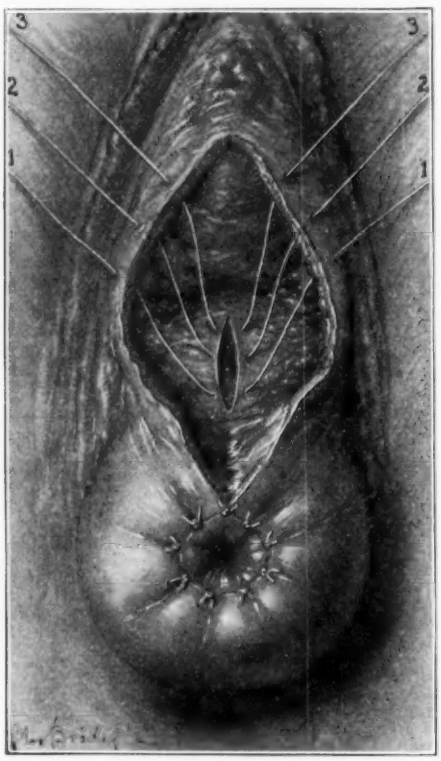


Fig. 8.—The vaginal mucosa has been attached to the cervix all the way around. The fixation sutures *1*, *2*, and *3* are now snugly tied and the intervening spaces approximated by a continuous suture.

The cervix was purposely left outside in order that the various steps can be clearly seen. At this point in the operation the cervix is actually well within the vagina.

dissected free. When this happens the opening is closed with fine continuous catgut, care being taken in the meantime to prevent any blood from escaping from the vagina into the peritoneal cavity.

The vaginal edges are now united to the cervix, as shown in Fig. 7. Figure-of-eight sutures here enable us to secure snug approximation with half the number of knots that would be necessary were interrupted sutures employed.

After the vagina has been sutured to the cervix, we have the picture shown in Fig. 8. The fixation sutures are now tied, giving us the picture shown in Fig. 9. As a rule, there is not an accurate approximation above and below sutures 3 and 1, and there is often slight gaping between sutures. To remedy this a continuous suture is used.

The perineum is now repaired.

The operation as shown above has given us excellent results and I know of no other plastic procedure in gynecology that is so univer-

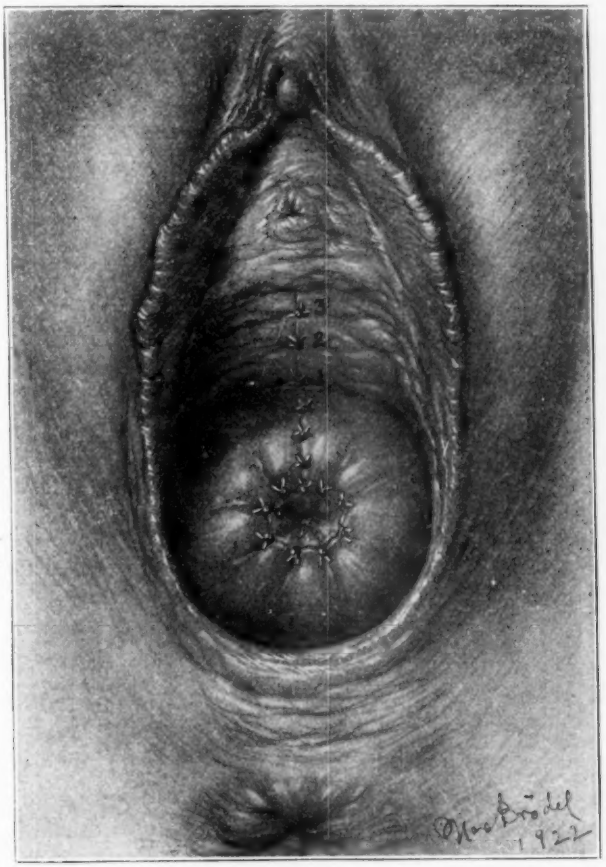


Fig. 9.—The cervix actually lies farther in than this. The perineum is now repaired and the operation is finished.
A catheter introduced into the bladder will show that it is now well lifted up. It rests on the fundus of the uterus.

sally satisfactory. I have been using the traction sutures for years. In a few cases in which the uterus has been exceptionally friable they have occasionally pulled out and it was necessary to introduce others. On the whole they are much more satisfactory than the tenaculum or mesoforceps. When we no longer need these sutures as tractors, they become the permanent fixation sutures.

20 EAST EAGER STREET.

*(For discussion, see page 567.)*

# PRE-CANCEROUS CONDITIONS OF THE CERVIX UTERI*

By R. R. Huggins, M.D., Pittsburgh, Pa.

WHEN one considers that an individual born today is entitled to live ten years longer than one born fifty years ago, we must realize how rapidly our methods of treatment are changing and will continue to change in the years to come. In a short time the offices of physicians will be filled with people who come to consult them with reference to the state of their health rather than wait until sick. They will want to know if they are up to their own normal and if their resistance is such that they will be able to withstand the invasion of infecting organisms and the strain incident to the stress of life. In other words, we have entered the stage of prevention and it marks a new era in the progress of scientific medicine. The subject and contents of this paper are so elementary that it seems like wasted energy, were it not somewhat doubtful that enough is said about the prevention of cancer of the cervix. It would seem that it is the duty of the members of this organization to teach more actively both the public and physician what they know about the prevention rather than continue to stress the symptoms. While the early symptoms are important, yet how seldom are we consulted at the time when the patient can be saved by any known method of treatment. In teaching prevention, we will educate the public to the necessity for heeding the early signs.

One of the most gratifying things in medicine is the wonderful progress made during the last twenty-five years in the prevention of disease and reduction of mortality. It is rather remarkable that while there is a steady reduction in the mortality of tuberculosis, there has been a corresponding increase in cancer. The reduction in the mortality of tuberculosis and many other diseases had been due largely to preventive measures and to a more intelligent appreciation by both the public and the profession of the underlying causes. Because no definite cause has been assigned to cancer and because of its frightful mortality, we have been somewhat slow as a profession in asking the public to share with us the responsibility of more active measures in its prevention. We hesitate to mention the word cancer to our patients for it carries terror equal to that of the electric chair. It would seem that the time has arrived that we should take our patients into our confidence and tell them all we know about this dread disease. Before doing this, however, the profession itself must

*Read at the Forty-seventh Annual Meeting of the American Gynecological Society, Washington, D. C., May 1-3, 1922.

appreciate a few important principles in the consideration of this subject.

It seems apparent that in injuries and irritations we have causes which promote the formation of malignant growths, but after all is said, there is a gulf between the cells of normal tissue and the cells of tumors which remains hidden and unexplained. The absence of any explanation should not blind us to the existence of certain clinical facts that are of great importance in a very practical way. Virchow believed that irritation was an important factor in the etiology of cancer, but it remained for Fibiger to produce carcinoma

Fig. 1.—P-1699-14. This figure illustrates the effect of the closure of the duct of a cervical gland by an overgrowth of squamous epithelium, the result being a retention cyst or Nabothian follicle. These cysts probably have no direct connection with carcinomatous change but serve as sources of chronic irritation and are associated with chronic cervicitis.

in the stomach and esophagus of rats by infecting them with spiroptera, the cause of the malignant change being irritation of the mucous membrane by the parasite. Yamagiwa after several years of experimental work on this subject concluded "that repetition or continuation of chronic irritation may cause a precancerous alteration in epithelium previously normal; if the irritation continues its action, carcinoma may be the outcome even though no specific agent has been interpolated." In a series of experiments carried on over a period of four years by Yamagiwa and Ichikawa in the production of carcinoma, painting with coal tar was found to be most effective. They

were able by repeated applications of coal tar to the ear of rabbits over a period of from thirty to three hundred and sixty days to produce new growths, which under the microscope showed carcinoma in its earliest stage. Certain of the growths continued to increase in dimension until they imitated even macroscopically the carcinomata of man. Cases of metastasis were demonstrated. They were able to produce eight cases of carcinoma in its earliest stage, sixteen in an early stage and seven complete carcinoma. The carcinomatous change was discovered between the fifty-fifth and three hundred and sixtieth day. In most cases it was found after the one hundred and

Fig. 2.—P-1699-14. The arrow in this figure points to a cervical gland which is lined with squamous epithelium instead of the normal columnar type. The gland probably ruptured and became relined by an extension from the surface epithelium. Bits of epithelium cut off in this way have been shown to be more or less directly associated with carcinomatous changes and are similar to the epithelial displacements in the pylorus of the stomach on which so much stress has been placed at the Mayo clinic. Other retention cysts with acute inflammatory exudate about them are seen in the section.

fiftieth day. They concluded that the continuation of chronic irritation may cause a precancerous condition in epithelium previously normal.

In general we are concerned with two factors, a predisposing cause and an exciting one. It is evident that the study must begin in the individual living cell and it seems probable that the predisposing cause is a chemical one, because tissue does not react the same to all forms of irritants. This has been proved in the study of occupational cancer. For instance among workers exposed to soot, which

is less irritating when compared to tar, coal dust, gas tar, pitch or blast furnace pitch, we find the greatest cancer incidence. Cancer is more frequent in organs exposed to fluids undergoing bacterial decomposition with its consequent chemical change, such as the stomach, rectum and cervix. The constant bathing of a chronic erosion of the cervix with the accompanying inflammation in a purulent acid secretion must be an important factor in the cause of malignancy. The period of time over which this may occur is especially important and should be given due consideration.

We must acknowledge that we do not know the exact cause of this

Fig. 3.—P-1788-14. This figure illustrates a displacement of squamous epithelium upward into the cervical canal and the extension of the epithelial tissue over the mouth of the gland resulting in cystic distention and chronic inflammation. This epithelial mass, while irregular and thick, is, to all appearances, nonmalignant. Such processes of squamous epithelium which extend upward into the cervical canal represent the opposite of the condition known as the erosion of the cervix which consists in a displacement of columnar epithelium downward onto the external surface.

peculiar change in tissue. We know that for some reason, the growth we call cancer, is a proliferation of cells previously normal, which increase in number until a mass is formed which attracts attention as a result of the changed condition of the normal structures. This does not occur in a day or month, but clinical experience teaches that it is a slow growth and that it probably has its origin in a single cell which has renounced its allegiance to the normal body control. How long the cause may have existed before the onset of malignancy

is unknown. We do not know whether certain systemic changes may predispose, or whether it is entirely local. If we have learned anything about the cause which is in any way helpful, it must be that constant irritation with its attendant inflammation plays a most important part in its development. The fact that it occurs almost always in tissue that is located in the body where there is more or less constant friction or irritation from the normal performance of function seems sufficient proof that this factor plays a most important part. This is sufficiently appreciated by all, so that farther dis-

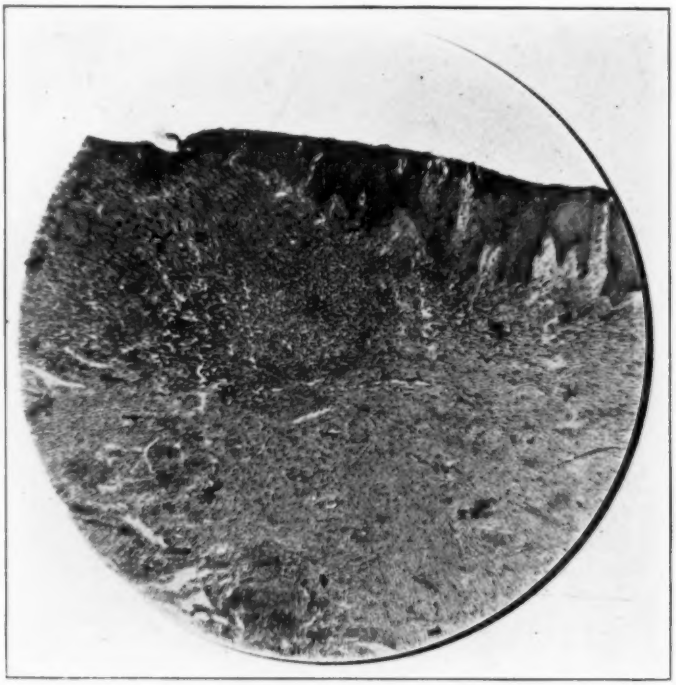

Fig. 4.—S-21-736. This figure illustrates the overproduction of squamous epithelium which has taken place in the repair of an actual cervical ulcer. There is no definite basement membrane present. The epithelial tissues have extended for a distance of two or three times further into the stroma of the cervix than happens under normal conditions, and present a ragged and irregular border. The cells are more or less atypical and the condition is definitely a precancerous one.

cussion upon this point is unnecessary. We can seldom anticipate when the cells may rebel, and a mutiny ensue with the result of malignant change but, if on the alert, we can see that the patient's general health is up to the normal and that all points subject to local irritation are kept free from inflammatory change. If this is true in other organs, then it must be equally true in the cervix. Our results so far as treatment is concerned in cancer of the cervix are very poor. Unless discovered and operated upon very early in the course of the disease the outlook is grave, and even then the operation

is accompanied by a considerable primary mortality, if radically performed. We may continue to emphasize the necessity for early diagnosis, but unless we are able in some way to prevent or anticipate the diseased change, we will be usually too late. It is true that many women do not consult a physician until the disease is well advanced, but practitioners are very slow in the serious consideration of symptoms almost pathognomonic of cancer of the uterus.

Just as we remove warts or moles or pull a tooth with sharp edges or excise an ulcer of the stomach to prevent the development of cancer, so we must learn to treat the cervix in all women approach-

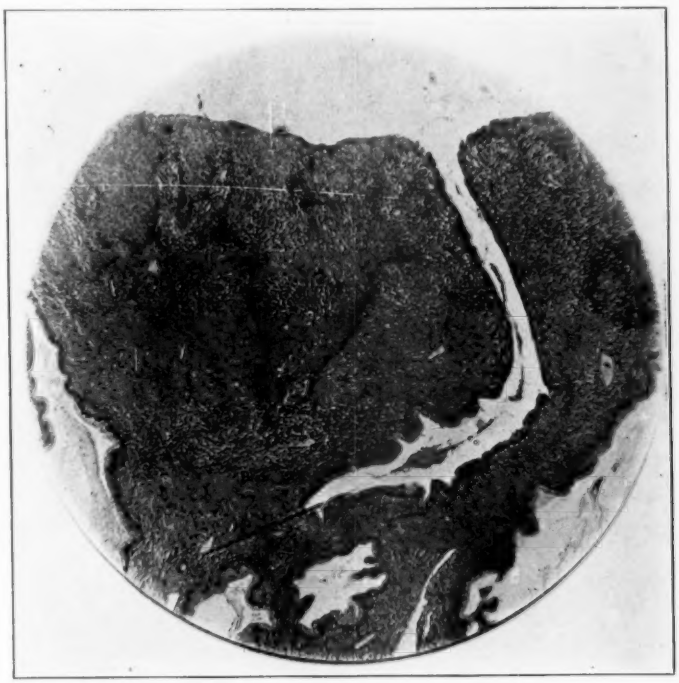

Fig. 5.—P-80-12. This figure shows the point of change between the stratified squamous epithelium and the columnar epithelium of the cervix. In this instance the squamous epithelium has undergone an atypical hyperplasia, is penetrating deeply into the stroma, is free from basement membrane and shows definite early malignancy. It is associated with chronic endocervicitis and hyperplastic and cystic cervical glands.

ing the age of forty. This is true, of course, more especially of the woman who has borne children. It is remarkable how seldom we find a healthy cervix in women who have given birth to one or more children. There is always chronic inflammation present where there is erosion and cyst formation incident to this process. It is quite likely that scar tissue in old lacerations is of little importance without the inclusion of the above mentioned conditions. So frequently do we find the cervix filled with cysts which are really due to a replacement of the epithelium. If we consider what happens in the healing

of an erosion it is perfectly easy to understand how the cervix becomes filled with these small cysts which cause a certain amount of irritation.

Erosion used to be called an ulcer, that is a loss of tissue; but on the contrary in erosion there is an increase of tissue and the condition should really be described as a sessile adenoma. The vaginal portion of the cervix is normally covered over with many layers of a squamous epithelium continuous with that of the vagina and at or about the os externum the epithelium suddenly becomes cubical and is in fact the columnar glandular epithelium which lines the cervix and the body of the uterus and which consists of a single layer of tall cells.

Fig. 6.—P-124-12. This figure illustrates an early squamous cell carcinoma of the cervix with hyperplasia of the epithelial layer and penetration into the stroma. At several points, epithelial masses are found extending into the lumina of the glands.

These cells are continuous with the glands of the cervix which are numerous and which are also lined with a single layer of columnar epithelium. The glands of the cervix are more complicated and larger than the glands of the uterus. In fact the cervix is itself a gland and is a structure quite apart from the body of the uterus. It is in connection with these glands or at all events with the columnar glandular epithelium lining of the cervical canal that our interest lies in the pathology of erosion. This red patch which is so typical in its appearance is due to an overgrowth and hypertrophy of the columnar cells lining the cervical canal which spread over and grow through the

squamous epithelium which normally covers the vaginal portion of the cervix. This bright red area then is not due to any destruction of tissue but simply to a replacement of deep columnar cells over a wider area than normal. The bright red color is due to the fact that the surface is covered by only a single layer of cells allowing the bright color of the underlying blood vessels to show through. If a return to the normal occurs it comes from a recovering of this surface by the normal squamous cells of the vaginal mucous membrane. When this happens these deep cells, glandular in type, become buried and covered over. Their natural function being to secrete, it is easy to

Fig. 7.—P-2359-15. This figure illustrates an adenomatous polyp of the cervix associated with severe chronic inflammation. There is no evidence of malignancy in this section, but the rather rare condition of adenocarcinoma of the cervix occasionally starts in this type of growth.

account for the formation of cysts. Masses of these little mucous cysts may occupy the surface of an erosion or extend deeply into the tissue of the cervix. The marked tenderness so often present in this condition indicates the presence of inflammatory change.

The presence of these cysts scattered throughout the cervix is common and the extent is often not appreciated until amputation or incision reveals their presence.

Every woman who has borne children and who has reached the age of forty should be examined and if disease of the cervix is found it should be treated. This treatment may be palliative but often it

should consist in the removal of the diseased tissue. We must remember that cancer starts in a small way, perhaps in one single cell. That while a cervix may present but slight evidence of disease, this may be sufficient in the presence of susceptibility and an acid medium to cause cancer. It is important that every practitioner acquaint himself with what a normal cervix is, so that he may appreciate the deviations from the normal and cure the cancer before it starts, because there is no doubt that this is our only means of dealing with this horrible disease at the present time.

Sixteen years ago I read a paper on this subject and reported four cases where cancer of the cervix developed in patients who had been under my care before its onset, and where previous examination had been made showing marked pathologic change in the tissues of the cervix, chronic in character, which undoubtedly predisposed to and preceded the malignant change. There has been no change in our ideas and all the evidence which has accumulated points to the following facts which were enumerated at that time.

First: That the cancer begins as a benign growth.

Second: That there is a true precancerous stage in which removal is a sure means of relief.

Third: That the disease is absolutely local in its beginning.

Fourth: That there is a varying degree of malignancy, some growths tending to return more readily than others.

Fifth: That as a rule tissues are more susceptible to its development at the age where atrophy and degeneration take the place of the building-up processes.

Sixth: That it is prone to develop in an acid medium such as the stomach or vagina.

I desire to acknowledge my appreciation to Dr. S. R. Haythorn and Dr. H. H. Permar for their help in preparation of the illustrations.

1018 WESTINGHOUSE BUILDING.

# Society Transactions

## AMERICAN GYNECOLOGICAL SOCIETY

FORTY-SEVENTH ANNUAL MEETING

*WASHINGTON, D. C., MAY 1, 2, 3, 1922*

(*Continued from October issue.*)

DR. JOHN A. SAMPSON, of Albany, N. Y., read a paper entitled **The Life History of Ovarian Hematomas of Endometrial Type.** (For original article see page 451.)

### DISCUSSION

DR. J. WHITRIDGE WILLIAMS, BALTIMORE, MARYLAND.—Dr. Sampson has done a great service in showing us three things. First, the origin of ovarian hematomata which, in the past, has been a great conundrum. Second, he has demonstrated the mechanism by which certain adenomyomata of the structures posterior to the uterus may develop. Third, he has proved the possibility of the transplantation of tubal and uterine epithelium upon other parts of the body and its subsequent growth.

I think no one who has seen these specimens can have any doubt about the nature of the ovarian hematomata which have been demonstrated to us. On the other hand, I do not feel that the arguments presented concerning the implantation of tubal and uterine epithelium are quite so convincing. But when I saw all of these specimens and talked with Dr. Jacobson I was convinced, so that I do not hesitate to tell you that I believe that everything Dr. Sampson has said is entirely justified. Dr. Jacobson experimented with rabbits, and after incising one horn of the biocornuate uterus, scraped off a little mucosa and imbedded it beneath the peritoneum in various parts of the body. Upon opening the animals weeks or months later he found small cysts at the site of implantation, some having the distinctive features of tubal, and others those of endometrial cysts. In other words, he was able to produce experimentally exactly the same condition that Dr. Sampson has shown us. Accordingly, I believe that the combination of Dr. Sampson's clinical observations with Dr. Jacobson's experimental work clinch the matter, so that I have no hesitation in endorsing everything Dr. Sampson has said, and I congratulate him on his splendid work, and the Society on having so enthusiastic a member, who is capable of conducting such investigation.

DR. JOHN G. CLARK, PHILADELPHIA.—Since Dr. Sampson brought out his paper last year we have been on the alert and have discovered three or four such cases as he has reported. This discovery of Dr. Sampson opens our eyes as to its possible relationship to the origin of adenocarcinoma of the ovary. The etiologic deductions are also of interest and I cannot but wonder if this misplacement of epithelium may have any bearing upon the etiology of the adenocystomata of the ovary. As to my personal observations I have held that these large cysts originate from inclusions of the glandular structures of the wolffian body. Certainly through

his painstaking search for new truths, Dr. Sampson has added a valuable discovery to the list of pathologic lesions of the generative organs.

DR. CURTIS F. BURNAM, BALTIMORE, MARYLAND.—Dr. Thomas S. Cullen has asked me to report three cases of adenomyoma of the abdominal incision. One of these cases followed an extensive operation for adenomyomatous uterus and adenomyoma of the rectovaginal septum eight years after the original operation. The other two cases followed ordinary cesarean sections, one of them belonging to Dr. Albert L. Stavely, of Washington and one to Dr. Ernest A. Codman, of Boston. The first case shows that it is possible to transplant adenomyoma; the other two indicate that ordinary uterine mucosa implanted into an abdominal incision can grow into a tumor exactly like adenomyoma. I may mention that all of these cases have been histologically studied and all are typical adenomyomata.

I have been much interested in what Dr. Sampson has said and in the comments of Dr. J. Whitridge Williams, because I remember two years ago talking with Dr. Sampson when his work was comparatively new. I did not see his specimens then and I will have to see them before I am convinced that the blood cyst of the ovary is analogous to or the same condition as adenomyoma of the uterus. We must remember that the surface epithelium of the ovary is ciliated and that it is quite possible for ciliated epithelium to grow down into the lutein or blood cyst after rupture. The cells which Dr. Sampson speaks of as endothelial phagocytes are very similar to the cells which we have spoken of as external theca or lutein cells.

DR. BENJAMIN P. WATSON, TORONTO, CANADA.—I would like to ask Dr. Sampson with reference to the stroma surrounding the glandular structures which he finds in the ovaries and other places. Since Dr. Sampson's first communication we have tried to investigate all ovaries containing hematomata, and our difficulty has been that while occasionally we found glandular epithelial structures, we have never been able to satisfy ourselves with regard to the stroma surrounding these glands, and it seems to me, the stroma is the essential part of this theory. We do not have menstrual bleeding except in the stroma, and this cannot arise from epithelial implants. Does Dr. Sampson believe that the stroma can be derived from these epithelial implants, and if not, how does that stroma, which will allow of menstrual bleeding, come about in this situation? That to my mind is the difficulty in accepting epithelial mechanical implantation occurring after middle life as an explanation of these conditions.

DR. WILLIAM C. DANFORTH, EVANSTON, ILLINOIS.—I would like to ask Dr. Sampson whether these cases can be dealt with in any other way than by total removal.

DR. SAMPSON (closing).—As I understand Dr. Clark's question it is this: When small implantations are found on the ovaries at operation, what shall be our treatment of this condition? My reaction at the present time is to be conservative of ovarian tissue, excising these areas without removing the ovaries if ovarian conservatism is desired. In cases with a hemorrhagic cyst and perforation, both primary implantation adenomas from the tubes and secondary ones arising from epithelium escaping from the perforated ovarian cyst, may be present. If extensive implantations are found I believe that all ovarian tissue should be removed.

Three of the cases in which I have done conservative ovarian resection, were not relieved by the operation. In one of these I operated a second time and found that the implantation adenoma had increased in size. Conservative ovarian surgery is a dangerous experiment in cases in which there is extensive implantation.

Dr. Watson asked about the origin of the stroma about the glands in these aden-

omas. In undertaking this work the first thing I tried to do was to obtain a knowledge of the uterine and tubal mucosa under different conditions. I have accepted as my standard of comparison the glandular elements found in primary adenomyoma of the tube and of the uterus, because we appreciate the fact that these growths have arisen from the invasion of the walls of the tubes and the walls of the uterus by the mucosal lining. We usually find in primary adenomyoma of the tube that the stroma so characteristic of the endometrium, is either absent or scanty. We will also find, if we study the cases of primary adenomyoma of the uterus, that as the growth begins to invade the uterine wall, glands without stroma may invade the uterine wall first and subsequently that stroma is developed. Furthermore, in the study of implantation adenomyomas of endometrial type the growth on the surface may not be accompanied by stroma, but after it invades the tissues of the organs beneath it the characteristic stroma of the endometrium develops around the glands and tubules. This would suggest that the stroma in these cases is derived from the tissue invaded and is not carried with the epithelium. One of the most convincing arguments in considering that these adenomas on the surface of the ovary are acquired and not congenital, is the fact that we find small (primary) implantations of apparently the same age and structure not only on the surface of the ovary but also on the peritoneal surface of the pelvis. Furthermore, they are most frequently found on the structures which come in contact with the fimbriated end of the tube.

In answer to the question of Dr. Danforth, my tendency is to be conservative from an operative standpoint in the cases of young women unless the growth is extensive. The majority of the cases of implantation adenomas are only of histologic and pathologic interest. Sometimes they acquire great clinical importance when they combine invasion (malignancy) with that of function because the menstrual activity gives rise to the most distressing symptoms.

DR. WATSON.—I would like to ask Dr. Sampson how he explains the hemorrhage in those cases of small tubes where there is no stroma. I have seen it present in some specimens apparently nontypical of endometrial stroma.

DR. SAMPSON.—We have found in most of these cases a little bit of stroma. I look upon the presence of the stroma not as absolutely necessary, but of importance in the development of the hemorrhage.

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PROFESSOR W. L. WILLIAMS, of Cornell University, by invitation, read a paper entitled **Some Phases of Bovine Genital Infections of Possible Interest to the Medical Profession.** (For original article see page 513.)

DISCUSSION.

DR. N. SPROAT HEANEY, CHICAGO, ILLINOIS.—Dr. Williams' experience as an obstetrician and gynecologist in veterinary medicine is much more extensive than the experience of any one of us here. His patients number thousands, while ours number hundreds. Dr. Williams is able to come to definite conclusions regarding the problems in which he may become interested. If he were to become interested in a subject such as Dr. Peterson spoke about last night, "Tuberculosis of the Genitalia," he could go to the slaughter house and by examination of the material at his disposal, find out what percentage of apparently healthy animals have tuberculosis of the pelvic organs. Or he could go to the slaughter house where known tuberculous

animals are butchered and find out what ratio exists in animals known to have tuberculosis between the lesions in the pelvis and those in the lungs.

Unfortunately, cows do not have cancer of the uterus or malignant tumors of the pelvic organs, so they are not suited to a very extensive study of malignancy. However, cows do have extensive inflammatory changes in the pelvis. Sterility in cattle will teach us much regarding our problems of sterility in the human family. Their period of gestation is similar and many interesting analogues may be found.

The value of cooperation between the biochemist and the clinician was mentioned a number of times. If, in our medical schools, we had a veterinary department, it would undoubtedly be very valuable. For instance, a veterinarian might tell us what animals are particularly suited to the particular problems we wish to study and would hardly recommend the gestation period of guinea pigs as being very helpful in the study of problems of gestation in the human family. The veterinarian is very exact in his information. During a pelvic examination, for instance, he can tell the difference between a corpus luteum and a graafian follicle. He can also tell from this examination just when the animal will again be in heat.

DR. J. WHITRIDGE WILLIAMS, BALTIMORE, MARYLAND.—I want to acknowledge to Dr. Williams my debt of personal gratitude for the two books he has written. A number of years ago he wrote his book on veterinary obstetrics, and when I bought it I read it from cover to cover, and last year, when he gave me a copy of his last book I read it with such extreme interest that I volunteered to write a review of it. In that review I stated that his work and that of many veterinarians was a revelation to me, and that they were doing many things which human gynecologists and obstetricians could but do not do, and they were obtaining a degree of precision in many things that we lacked.

I could relate many features of veterinary obstetrics because they have interested me profoundly. My original interest was aroused by studying the placenta of the cow, in preparation for a course on comparative placentation. Since that time I have been particularly interested in another disease which Dr. Williams did not mention in his paper, but which is of extreme interest, namely, parturient paresis. As you know, the disease nearly always affects a perfectly good cow, who, shortly after she has a calf, lies down, sticks out her hind legs, is unable to get up and passes into a condition of coma and dies, with albumin and sugar in the urine. In recent years a method of treatment has been introduced for this disease which cures them all, and consists in squirting air into the udder by means of a bicycle pump and in a few hours the cow gets up and walks away cured. The treatment is so universally successful that only occasionally a cow dies, and for several years I have had veterinarian friends looking for cows who had died of the disease so that I could get autopsies. I have not yet succeeded.

Cattle present extraordinarily interesting problems. For instance, Dr. Williams could tell you about the venereal diseases in cattle and it would surprise most of you to know that practically every cow suffers from them.

DR. WILLIAMS (closing).—I do not think I have anything to add except to express my appreciation for the attention and interest which my paper has elicited, and to voice my belief in the value of the recognition of veterinary and human medicine as one science. I hope that in their purely scientific aspects they will become more and more unified as time goes on.

DR. EDMUND B. PIPER, of Philadelphia, Pa., by invitation, read a paper entitled **The Treatment of Puerperal Sepsis with a Report of Animal Experimentation in the Chemical Disinfection of the Blood by the Use of Mercurochrome.** (For original article see page 532.)

DISCUSSION

DR. BARTON COOKE HIRST, PHILADELPHIA, PENNSYLVANIA.—One of the most disappointing things in modern medicine is our failure to make progress in the treatment of blood infections by septic microorganisms. Other scourges of mankind are more amenable to treatment than they were forty years ago. I sent recently a questionnaire to the chief centers of the country and was amazed at the lack of initiative shown by the replies, in this work. Dr. Polak alone had something to suggest. He advocated repeated transfusions of small quantities of blood. In none of the other centers was anything being done to advance our knowledge of the treatment of puerperal sepsis. Dr. Piper's work shows at least an attempt to improve our means to deal with the problem. It is creditable clinical study with hopeful results, and a very pretty piece of laboratory work. I have observed the treatment of puerperal sepsis for a long time, with unusual opportunities because I believe the maternity of the University of Pennsylvania is one of the few places in the United States which solicits these septic cases from the outside for study. What is also important, my state of mind has been skeptical in watching these new propositions because I have had so many disappointments, but after a careful observation of Dr. Piper's clinical application of his experimental work, I am convinced that this form of chemical disinfection of the blood must have a place in the future treatment of blood infections by septic microorganisms. Dr. Piper could not give the details of the cases in which this treatment was successful, but I had the opportunity to study these patients from day to day, and I am convinced that with this form of chemical disinfection of the blood we can effect temporary improvement at least, allowing time for the ultimate establishment of immunity. For example, in a blood giving numerous colonies in culture experiments in twenty-four hours, it would take five days after one of these treatments to get a positive blood culture with but few colonies. Coincidentally the patient would be distinctly better and so on until immunity was established. Some of these cases would, I believe, have died without this aid.

My conviction is that we have in this method of chemical disinfection of the blood a valuable aid in the treatment of blood infections by septic microorganisms. It is, therefore, incumbent on us, I think, to give these patients the benefit of this treatment if we are to render them every aid possible; not as a specific, but in addition to fresh antistreptococcic serum, demonstrated in my clinic to have distinct, and sometimes brilliant results, and to all the other methods of treatment that have been shown to have some efficacy.

DR. BROOKE M. ANSPACH, PHILADELPHIA, PA.—I have been much interested in these experiments of Dr. Piper, having known of them from the beginning, and especially so in view of the work which was presented before this Society at the last meeting in Philadelphia by Chalfant and Miller, of Pittsburgh, relative to the use of arsenobenzol in hemolytic streptococcus blood stream infections. At that time in the discussion of the paper, Dr. Hare, of Philadelphia, pointed out that no experiments had yet proved that arsenobenzol would kill the spirochete pallidum in a test tube, and that Ehrlich himself had realized there was some reaction after the introduction of arsenobenzol into the blood stream which actually destroyed the germ of syphilis. He further went on to say, that whereas the spirochete belongs

to the animal kingdom, hemolytic streptococci belong to the vegetable kingdom, and Ehrlich did not claim any effect upon the latter. Hare suggested laboratory experiments to clear up these questions. Whether such have been made by others or not, I do not know, but if I understand him correctly, Dr. Piper has shown that mercurochrome can kill, or seriously impair the activity of the streptococcus hemolyticus when suspended with it in blood serum.

I think there is a distinct promise in the work of Dr. Piper of an addition to our therapy in combating these severe infections. The mere fact that the use of a chemical alone has not cured the patient is no argument against its efficiency in one direction at least. Is it not likely, in these severe blood stream infections, that although we may destroy the streptococcus with a germicide, we do not get rid of the toxin, and that is probably what kills the patient?

I have been told by men who direct the preparation of the sera in one of the large and well-known manufacturing companies of the country, that the results from the use of sera in artificially produced infections were amazing. Some time we will be able to do the same thing with the human subject. May it not be hoped that by this combination of the use of antiseptics, such as mercurochrome, to kill the bacteria, and the injection of antistreptococcic serum or other sera to neutralize the toxins, a definite improvement in our therapy may be effected?

DR. JOHN A. McGLINN, PHILADELPHIA.—Piper's study takes us back to the time when formalin was employed in this type of case. Practically everything in the disinfectant line has been injected into the blood stream with the hope of curing these cases of blood infection. Perhaps Dr. Piper's assumption that it forms an autogenous vaccine by cutting off the bacteria is not so far fetched. It is not so much the antiseptic action of the bacteria in the blood stream as it is that a foreign protein is thrown into the blood stream. Cases of pneumonia have been treated by Dr. Fred B. Robertson by the use of paratyphoid injections given intravenously. Recently we have been using foreign proteins, such as milk, in pelvic infections, particularly a generalized infection, and the results are amazing. Patients have a severe reaction, the temperature ranging from 104° F. to 105° F., then dropping to subnormal. In one case a large pelvic mass entirely disappeared, the fixed uterus became freely movable, and after the second injection the mass disappeared and the pelvis was absolutely normal. It is more than a blood infection. The organisms are distributed by the blood, but there are foci in various parts of the body, and they are hard to get rid of in the short length of time the germicide remains in the system.

DR. ARTHUR H. CURTIS, CHICAGO.—We have gone through the milk and other foreign protein stage, and have not noticed a distinct change. I do not think the remarks that have been made should go into the proceedings without objection; foreign proteins appear to be momentarily helpful, but are evidently of no permanent value.

DR. PIPER (closing).—It is due to the kindness and consideration of Dr. Hirst that I have had placed at my disposal clinical material for what small amount of clinical work there has been done. There have been times during the past two years, when I have seriously considered dropping the whole matter. This has been due to the negative results that we got for a time owing to faulty technic.

I want to thank Dr. Anspach for his remarks and especially for what he said about arsenobenzol. Although we have not had any results from this drug, we do not mean to say that others may not. We do not know that the streptococcus is killed, but these patients definitely become clinically better. That is the only positive statement that can be made. The negative blood culture may or may not mean that the organisms have been destroyed.

Dr. McGlinn has called attention to the various attempts in years past with chemical disinfectants. The tests done with bichloride of mercury and formic aldehyde are probably the two best known examples. When bichloride of mercury was used, 30 minims of a 1:10,000 solution were given intravenously. We are using from 25-50 c.c. of a 1 per cent solution of mercurochrome, approximately 1,500 times stronger. Our effort has been to produce within the blood stream a dilution of sufficient strength to be germicidal to the streptococcus without being toxic to the individual.

It must be remembered that in all of these cases there is more than blood stream infection alone. We have no idea that we are presenting a cure-all. So far as the actual sterilization of the blood is concerned, no one feels the hopelessness of it more than I. Perhaps, the blood may be sterilized temporarily, but the original focus may keep pouring out fresh organisms into the blood stream. Our hope is, that we may sterilize the blood stream temporarily, and by repeated sterilization, if such a thing is possible, we may aid the human organism to gradually increase its resistance up to the point where it is able to throw off and conquer the infection itself.

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Dr. Thomas S. Cullen, of Baltimore, Md., read a paper entitled **A Point in the Technic of Vaginal Fixation of the Uterus.** (For original article see page 544.)

DISCUSSION

DR. J. RIDDLE GOFFE, New York City.—I had the privilege of seeing Dr. Cullen, in doing the interposition operation, apply the point of technic which he has just described. I was surprised at the difficulty he experienced in delivering the fundus of the uterus into the vagina preparatory to inserting the stitches which were to secure the fundus between the bladder and the vagina. It was a case of procidentia and the pelvic organs were easily reached through the vaginal incisions.

Later, I discovered, that after amputating the cervix and before attempting to deliver the fundus, he had attached a heavy traction forceps to the cervix, which held it at the vulva. This revealed the mystery, viz: the presence of the difficulty his procedure was designed to overcome; in order to have both poles of the uterus at the vulva at the same time it was necessary to flex the fundus extremely on the cervix, and this the uterus was not long enough to permit. I am sure the difficulty can be avoided if, in the process of delivering the fundus, he at the same time pushes back the cervix into the vaginal culdesac, instead of holding it at the vulva.

DR. THOMAS J. WATKINS, Chicago.—The tension sutures, illustrated by Dr. Cullen for delivery of the body of the uterus and for fascia fixation, it seems to me, will prove to be very serviceable. Any one who has done many of the transposition operations must have been impressed with the fact that in occasional cases the delivery of the body of the uterus is rather difficult, and that consecutive passage of the suture through the body and cervix of the uterus is also difficult in some cases. Dr. Cullen's sutures solve these difficulties.

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Dr. R. R. Huggins, Pittsburgh, Pa., read a paper entitled **Pre-Cancerous Lesions of the Cervix.** (For original article see page 552.)

*(To be continued.)*

# Department of Reviews and Abstracts

CONDUCTED BY HUGO EHRENFEST, M.D., ASSOCIATE EDITOR

## Collective Review

### Lumbar Puncture in the Treatment of Eclampsia

BY RAMSAY SPILLMAN, M.D., WASHINGTON, D. C.

THIS review is not a brief either for or against lumbar puncture in eclampsia. It is an attempt to find, in the literature, an answer to the question, "What happens when a lumbar puncture is done in eclampsia?"

The first to publish an account of the use of this therapeutic measure in this condition appears to have been T. A. Helme,[9] whose description of one case appeared in the *British Medical Journal*, May 14, 1904, four and one-half months before the appearance of Krönig's more extended observations[12] in Germany. Helme suggested "that the eclampsia is due to increased cerebrospinal tension" and was so gratified by the result of the puncture that he lamented having refrained from using it in a previous case presenting a similar clinical picture, which went on to a fatal ending. Similarly, Snyder[17] performed a lumbar puncture on an eclamptic patient who was in extremis, and the patient promptly took a turn for the better and recovered. However, such brilliant results are unfortunately not typical.

For this review, seventy cases have been analyzed and tabulated in a search for the answer to the following questions:

1. Is lumbar puncture a rational procedure?
2. Does it jeopardize the patient's chances of recovery?
3. Does it add to the patient's chances of recovery?
4. How much fluid is there precedent for withdrawing?
5. What is the prognostic significance of bloody fluid?

1. Helme's suggestion as to the rationale of the treatment was supported by the contemporary observations of Krönig and by many others since. Krönig accepted as the normal intraspinal pressure, 120 mm. of *water* (note: 13.5 mm. of water is equivalent to 1 mm. of mercury). The accompanying charts show pressures in eclampsia ranging from 120 mm. of water to 600.

Martin Fischer[24] in this country and Zangemeister[23] in Germany hold that the convulsions are due to edema of the brain. At all events, edema of the brain has been demonstrated repeatedly at autopsies on patients dying from eclampsia, and the amounts of spinal fluid withdrawn at lumbar puncture (see chart), together with the increased pressure, are in keeping with this condition. It is not the province of the present writer to express an opinion as to Fischer's work. It has been attacked

CHART SHOWING THE EFFECT OF LUMBAR PUNCTURES ON ECLAMPTIC CONVULSIONS IN 70 PATIENTS

by able men. Fischer's conception that the edema is not the result of nephritis but a concomitant of it, and his treatment of edema by the administration of salt and alkali are as far removed from medical orthodoxy as Martin Luther's pronouncement was from theological; but the results that Fischer reports demand consideration. "Edema and Nephritis" is a book which should be read before one either adopts or condemns Fischer's teachings. It may or may not lead one to conclude that there is a better way to reduce cerebral edema than by lumbar puncture, and renal glaucoma than by decapsulation.

2. Does lumbar puncture jeopardize the patient's chances of recovery? Cassagnes[6] concluded, from his collection of reports of fifty-five cases, that it is harmless. However, it should be borne in mind that there are two dangers from lumbar puncture: immediate and mechanical, and remote and infectious. Dandy[25] has recently said of lumbar puncture in brain tumor, "It has no useful purpose, but is accompanied by danger. I have seen at least four deaths following lumbar puncture, and many other patients have had distressing symptoms for many days. The danger lies in the sudden entrance of portions of the cerebellar lobes into the foramen magnum when the spinal fluid is released; the herniated cerebellum, being caught in the bony ring, compresses the medulla and upper spinal cord. Were there any benefits to be gained commensurate with the risk which is always present, lumbar punctures might be sanctioned." That the same dangers do not attend lumbar puncture in eclampsia, the present writer is not willing to assert. As for the remote danger, Weed, Wegeforth, Ayer, and Felton[26] have shown that lumbar puncture in the presence of an infection of the blood-stream invites meningeal infection, and one may or may not be certain that the blood-stream is free from infection at the time of puncture. Such a complication, of course, is a remote one in both senses of the word, but it is to be kept in mind.

3. Does lumbar puncture add to the patient's chances of recovery? In a few of the reported cases the prompt turn for the better certainly suggests cause and effect. Yet in one of Henkel's cases[10] a dry tap was followed by cessation of convulsions. One may add up the number of convulsions shown in the accompanying chart and note that 66 patients had more than 513 convulsions, an average of eight, before the puncture, and 508 convulsions after puncture, an average of eight. But I know of no way of telling how many convulsions these patients would have had without the puncture, and can only conclude that the average number of convulsions was 16, while DeLee in his text gives the average number as from five to 15. Of 68 patients treated by puncture, 19 died, a mortality of 27.6. The footnotes on the chart would indicate that in at least nine of these cases no treatment would have saved the patient, while a tenth died after 265 convulsions—though Spalding[22] has collected reports of 17 cases and adds one of his own, which had 70 or more convulsions, with recovery after 207, 265, and 500 convulsions, respectively. The maximum number he found was 593; this patient died.

In 25 cases out of 68, lumbar puncture was followed by complete cessation of the convulsions; yet in two of these cases there was a dry tap, which makes it questionable whether the canal was entered at all. Of these 25 patients, four died. Three deaths were due respectively to

streptococcus septicemia (this was a case with dry tap), pneumonia, and intraventricular hemorrhage.

4. As much as 101 c.c. of fluid have been withdrawn at one time, with recovery. From 20 to 30 c.c. is the amount that has most often been withdrawn, in the accompanying reports.

5. Prognostic significance of blood in the spinal fluid: Bar[2] found blood in the fluid and at autopsy found a large clot in the right lateral ventricle of the brain, and stated that bloody fluid bespeaks a fatal prognosis. However, in ten cases in this collection there were but four deaths, and in one case, frank hemiplegia with recovery, the fluid was bloody for three successive taps and clear on the fourth.

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# Selected Abstracts

## Pain Relief in Labor

**Hill: Anesthesia in Obstetrics:** The Ohio State Medical Journal, 1921, xvii, 228.

The author decries the fads in obstetrical anesthesia with their misleading claims for painless labor. He believes, however, that labor can be made comfortable by the proper use of anesthetics compatible with perfect safety to both mother and child.

The selection of the anesthetic should be determined by the individual case. The anesthetic best adapted to the type of labor and circumstances with which the accoucheur has to deal, should be the one of choice. Two general methods of pain relief are mentioned: (1) General anesthesia, for which ether or nitrous-oxide-oxygen are advised; and (2) analgesia or amnesia, for which morphine and scopolamine are used.

The first stage of labor is made comfortable by the use of small doses of scopolamine-morphine given after the pains have become regular. The danger

to the child is slight, provided the drugs are not given late in labor. In a primipara scopolamine-morphine should not be given after the cervix is three-quarters dilated.

The amnesia produced by these drugs during the first stage may be followed by nitrous oxide-oxygen administered with each pain during the early part of the second stage. As labor advances and pains become more severe, ether is advised. At the end of the second stage the patient is completely anesthetized and the head delivered by Ritgen's maneuver.

The author believes that ether may be administered much earlier in labor than is generally supposed. The use of a closed cone method of administration, such as the "Ben Morgan" apparatus, is recommended for this purpose. The use of chloroform is not advocated, having been abandoned by the writer ten years ago.

The routine use of drugs which stimulate uterine contractions postpartum is advised following general anesthesia, as a prophylactic measure against postpartum hemorrhage. NORMAN F. MILLER.

**Clarke: Anesthesia and Analgesia in Obstetrics.** Journal of the Kansas Medical Society, Oct., 1921, xxi, 313.

General observations on anesthesia and analgesia during delivery force the conclusion that women are entitled to relief from pain. Proper drugs help and do no harm. Standardization of methods for individual needs is not possible. The method used must give the greatest relief with the least danger to both mother and child. Hot enemas, bromides and chloral, morphine with scopolamine may be used in the first stage; nitrous oxide and oxygen, ether or chloroform in the second. Best results depend on the psychic control of the patient. The physician should not wait until the last minute to attend his patient.

W. K. FORSTER.

**Haultain: Further Experience of the Conduct of Labor Under "Twilight Sleep."** Edinburgh Medical Journal, 1921, xxvii, 27.

The author draws his conclusions from a series of over 800 labors, the last 150 are reported. Complete amnesia was obtained in 80 per cent of the primiparæ, in 50 per cent of the multiparæ. Partial amnesia occurred in 20 per cent of primiparæ, in 30 per cent of multiparæ, and short labors prevented the drug being effective in the remaining 20 per cent of the latter.

The method employed is as follows: The patient, her ears filled with cotton, is placed in a quiet, darkened room from which the relatives are excluded. When labor is fairly well established (pains occurring regularly every ten minutes in a primipara, earlier in a multipara) morphine gr. ¼ and hyoscine gr. 1/150 are given hypodermatically. Three quarters of an hour later and every hour after, the patient receives gr. 1/450 of hyoscine. To control restlessness morphine is used in the first stage, chloroform in the second. Chloroform is used when needed at delivery. On awakening from the sleep that usually follows delivery, the patient is unaware that labor has occurred. The author has found that large doses of morphine do not cause fetal oligopnea or asphyxia.

The largest number of doses of hyoscine given in any successful case was 53, the smallest 4. Although the patients may be restless, complain of pain or even be delirious, the end result is usually complete amnesia. Haultain thinks that the routine dosage of hyoscine is without danger, that these patients require less personal attention than those delivered in the usual way. An expert nurse

only is in attendance early in labor. The drug apparently shortens the first and probably delays the second stage somewhat. Forceps were used 41 times in the 150 cases. Three per cent of the babies were born oligopneic but needed no attention. Four babies were born asphyxiated, three after difficult forceps and one after delay with the aftercoming head. Scopolamine favors moulding in difficult cases. No dangerous complications were noted in the mothers. Two cases of mania were controlled with chloroform. The author believes that by removing the fear of subsequent labors, this procedure will increase the birth rate. The opposition to its general use resembles that seen at the time of the introduction of chloroform into obstetrics. H. W. SHUTTER.

**Zweifel: Painless Labor.** Muenchener Medizinische Wochenschrift, 1922, lxix, 52.

The author claims, from personal observation, that "Twilight Sleep" does not increase the birth mortality, and in a series of cases of twilight-sleep-children, followed for over ten years, there has been no evidence that this form of amnesia gives rise to any permanent injury. S. B. SHOLHAUG.

**Gerson: Twilight Sleep.** The Lancet, London, 1922, ccii, 428.

The author gives a résumé of his experience in conducting seventy-five confinements under twilight sleep. He kept very careful records of every sign and symptom, and all dosages were charted at the time given.

His technic in the case of a multipara was to commence the treatment as soon as labor set in. The first injection consists of 1/150 gr. scopolamine and 1/3 gr. morphine. The second injection of 1/150 gr. scopolamine was given in one hour's time, true amnesia generally being obtained three-quarters of an hour after this injection. The true state of amnesia was recognized by showing some familiar object to see if the patient recognized it. Analgesia was always complete regardless of the degree of amnesia.

The author found the use of forceps in the latter part of the second stage to be more frequent. In none of his cases could he attribute infant mortality to the use of drugs. Postpartum hemorrhages were in no appreciable way increased.

The author includes much valuable statistical data in his article as well as the results of his seventy-five cases. NORMAN F. MILLER.

**Horwitz: Twilight Sleep and the General Practitioner.** The Lancet, London, 1921, cci, 1154.

The author gives his opinion regarding twilight sleep based upon personal experience. He makes it a rule never to give the first injection until pains are coming regularly every five or ten minutes. In multiparae treatment is commenced as soon as the patient is definitely in labor.

The technic used by Horwitz is given in detail in his article. He believes the first stage to be definitely shortened and the second stage frequently prolonged and operative delivery more frequently necessary. The author has had but little difficulty from asphyxia of the newborn child in these cases.

The advantage of twilight sleep is emphasized especially in breech cases, and in patients with a minor pelvic contraction and those suffering from a heart disease. NORMAN F. MILLER.

**Opitz: The Alleged Danger of Twilight Sleep.** Muenchener Medizinische Wochenschrift, 1922, lxix, 261.

Of 4,279 births at the Universitaets Frauenklinik in Freiburg, 2,037 were conducted under "twilight sleep", and 2,242 without it. Among the former, 2.1 per cent were born dead or died within the first nine days; among the latter, however, the mortality was 3.75 per cent.

The author holds that in competent hands twilight sleep affords a protection rather than a danger to the child, but he does not consider it a procedure to be employed by the general practitioner. S. B. SOLHAUG.

**Lembcke: Comparative Study of the Action of Twilight Sleep with Modified Technic.** Muenchener Medizinische Wochenschrift, 1921, lxviii, 262.

The author compares the results (to both mother and child) obtained in his employment of morphine-scopolamine amnesia according to the "morphine-rich" technic of Siegel, with results obtained in the use of a modified "morphine-poor" technic. Essentially the difference is that in the latter the amount of morphine is reduced by one half, and the scopolamine increased by one-eighth over that of the classic procedure of Siegel. As concerns the mother, the modified technic gave amnesic results in 97.8 per cent of cases while the classic procedure yielded 92.0 per cent. With the modified technic 83.8 per cent of children breathed immediately on birth, 7.8 per cent before, and 8.3 per cent after the lapse of a minute, while with Siegel's technic the percentages were 38.3, 13.3, and 48.3 respectively. The mortality rate of Mayer is quoted who, deducting deaths obviously due to causes other than the scopolamine-morphine influence (such as placenta previa, contracted pelvis, premature separation of placenta, etc.) found that 1.1 per cent of children died as the result of birth trauma with the morphine-rich technic as compared with 0.5 per cent with the modified morphine-poor technic. S. B. SOLHAUG.

**Liegner: Labor Under Simplified Schematic Twilight Sleep.** Deutsche Medizinische Wochenschrift, 1922, xlviii, 424.

Liegner is opposed to the schematic method of twilight sleep as proposed by Siegel. He holds that no potent drugs should be given according to a fixed schedule, but that individualization is imperative in their administration. Even with the modified method, he has seen unmistakable evidence of retardation of labor, deleterious effect upon the mother and asphyxia and death of the fetus. This in spite of the reduction of the dosage of morphine and the addition of quinine.

He concludes that the administration of twilight sleep of any sort is out of question in general practice. Even under the most fortunate hospital conditions and with most expert attendants, he feels it should be employed only in exceptional cases and then only under the continuous personal supervision of the obstetrician. R. E. WOBUS.

**Schellekens: Analgesia During Labor.** Nederlandsch Tijdschrift voor Geneeskunde, 1921, ii, 2060.

After mentioning briefly the use of chloroform and ether in labor, the author reviews rather exhaustively the literature on twilight sleep, which brings him to the conclusion that the unpleasant effects on the mother and, especially the danger to the fetus, makes it an unjustifiable procedure. The pudendal anes-

thesia proposed by van Ilmer has had little support and has been generally discarded by those who have used it.

Sacral anesthesia, as first used by Cathélin, has given rather satisfactory results and has been recommended by various obstetricians, especially Stoeckel. Various operators have used solutions containing ½ to 2 per cent novocaine usually with sodium chloride or bicarbonate as well as adrenalin. From 20 to 30 c.c. are injected into the sacral canal while the patient lies with her hips slightly raised and the knees drawn up. After the needle is inserted, and before injecting the solution, the piston is withdrawn partly to exclude the possibility of injecting into a vein. Since all methods of analgesia have a tendency to weaken the pains, Schellekens made his injections only after the cervix was dilated and then noticed no effect on the contractions. If necessary, a further injection of 10 c.c. may be made. His results have been very satisfactory and he reports absolute analgesia in his cases, some patients experiencing merely a feeling of pressure as the head passes over the perineum. R. E. WOBUS.

**Gillespie: Relief of Pain in Labor.** Ohio State Medical Journal, 1921, xvii, 669.

The author is an advocate of chloroform as an analgesic for the country doctor who must meet all kinds of emergencies and cannot carry a complicated apparatus everywhere he goes. He gives chloroform only at the beginning of the pain before the patient is aware that the pain is coming. The patient takes three or four breaths and then is made to bear down the same as in the use of nitrous oxide. The article includes a discussion on how to administer chloroform in a normal delivery; handling difficulties in obstetrical anesthesia; objections to anesthesia in labor; anesthesia in the first stage; the prevention of septic infection and conditions foretelling difficult and tedious labor. This is an excellent article especially for recent graduates, who are familiar only with the adverse side of chloroform. W. K. FOSTER.

**Danforth: Nitrous Oxide-Oxygen Analgesia and Anesthesia in Normal and Operative Obstetrics.** The Pennsylvania Medical Journal, 1921, xxiv, 383.

The paper is based on about seventeen hundred obstetrical cases in which nitrous oxide-oxygen analgesia or anesthesia was used.

The author's technic consists in beginning the administration of the gas at the onset of the second stage. The mask is placed over the face and the patient instructed to breathe deeply and quickly, the number of breaths required varying between three and eight, depending on the individual. It is essential for the best results to begin the administration of the gas immediately at the onset of a pain. The use of oxygen with the nitrous oxide is advised in percentages from five to fifteen according to the individual requirements. Cyanosis should be avoided at all times. With the end of the second stage a little ether is added to advantage.

The course of labor is not slowed by a properly given gas analgesia, on the contrary the effectiveness of each contraction is increased because of the marked relief from pain and the voluntary cooperation of the patient. The danger of postpartum hemorrhage is not increased. The nitrous oxide does not relax the uterine musculature to the same degree as other anesthetics. No untoward effect of the gas was noticed in the infants and in no instance could the nitrous oxide analgesia be charged with causing fetal death.

For various operative procedures as repair of the perineum, low forceps, induction of labor by the introduction of a hydrostatic bag; and in eclamptic or

toxic patients where ether or chloroform would have a deleterious effect upon the parenchymatous organs, nitrous oxide-oxygen anesthesia, aided by a little ether if necessary, proves very satisfactory.

In version cases ether anesthesia is advised.

With an experience extending over a period of five years, the author concludes that a certain technic is essential in administering the gas to obtain the most successful results. Further he believes the value of nitrous oxide-oxygen in relieving pain in normal labor, may be extended to many obstetrical operative procedures, provided the anesthesia be given with a reasonable degree of skill.

NORMAN F. MILLER.

**Sellers: The Use of Nitrous Oxide—Oxygen Analgesia and Anesthesia in Obstetrics.** New Orleans Medical and Surgical Journal, 1921, lxxiv, 109.

Nitrous oxide is advocated as being the nearest to an ideal anesthetic that we have at present because "it is inhaled like air; is practically odorless; is almost instantaneous in its action on the patient; is eliminated so quickly that the gas has left the body by the time the next pain is due; can be used for every pain." Chloroform and ether nearest approach nitrous oxide as analgesics but they are not quickly eliminated and have many other disagreeable features. Nitrous oxide-oxygen shortens the second stage, gives relaxation, does away with restlessness and rigidity in most cases, decreases exhaustion, allows a rapid return to normal for the mother and does not affect the baby.

The patient should be assured that the pain can be relieved. When the pain comes, give enough to relieve it so that the patient will have confidence in the procedure. Determine just the amount necessary for each individual patient, usually two to four breaths and hold the last one. Start the inhalations at the first signs of the approaching pain. It is concluded that nitrous oxide analgesia is an almost ideal anesthetic agent in obstetrics and makes the best anesthetic for operative obstetrics. Ether is the next best agent and chloroform and twilight sleep should not be used.

W. K. FOSTER.

**Vignes and Moreau: The Analgesic Action of Nitrous Oxide on Uterine Pains.** Presse Médicale, 1921, No. 24, p. 234.

Following the work of Lynch and Heaney, the authors have investigated the use of gas in eight primiparæ and one multipara during labor. They concluded that there was no toxic effect during administration either for mother or infant. The deliveries proceeded in normal time and manner. The gas seemed to have little effect on uterine contractions.

F. L. ADAIR.

**Oettingen: Childbirth in Hypnotic Twilight Sleep.** Muenchener Medizinische Wochenschrift, 1921, lxviii, 265.

This is a report of a series of 16 women delivered at term under the influence of hypnotic suggestion, 14 of which were successful. The writer discusses the physiology of hypnosis and treats the subject historically. His treatment is begun three weeks before term; three or four women are hypnotized together, the power of suggestion being thereby strengthened. The suggestion is made that sleep can be regulated at desire, and that during sleep all pain ceases. Women coming in for delivery, actually in labor (pain), go to sleep very easily. A slight moaning or body movement during the pains reveals that labor is in progress. In the second stage, while patients do not reach the degree of excite-

ment experienced in scopolamine-morphine twilight sleep, they become restless and sometimes require three or four grams of paraldehyde rectally to deepen the sleep. Complete loss of memory or amnesia was achieved in six cases, partial amnesia in eight, with the stress of memory laid on inconsequential detail rather than on any pain experienced. The writer considers this an ideal adjunct to the conduct of labor and one that can be employed by any one giving it sufficient thought and attention.

S. B. SOLHAUG.

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# Book Review

**Gynecological and Obstetrical Pathology.**—ROBERT TILDEN FRANK. D. Appleton & Co., New York, 1922.

In the preface, the author states that he has attempted "to co-relate information of interest to the clinician and pathologist in such a fashion that both will derive full benefit from this conjunction, and in the first paragraph of the introductory chapter he lays down the following important rule: "Cooperation between the clinician and pathologist is necessary to obtain the most useful results. The pathologist ought not to be a stranger at the bedside or in the operating room. The clinician should prove a welcome guest in the laboratory and at the autopsy table."

With these aims in mind of the author, and with his personal knowledge of his ability, the reviewer began his task with the most pleasant anticipations, but regrets to state that after a careful perusal of the book they were not entirely fulfilled. This is due in part to three main factors: First, that in attempting to make the work attractive to the clinician, a considerable amount of information irrelevant to the pathologist has been introduced, which impairs its scientific value. Secondly, the style is defective, in that it often lacks in clearness, and thus makes it difficult to distinguish between what is important and what is not. Moreover, the work suffers from a too casual or jocular tone, as, for example when the author speaks of the "occasional gynic operator" or states that "derivatives of the three layers of the embryo are arranged in a more or less orderly fashion in dermoid cysts, and scrambled together in a 'potpourri' in the solid teratomas." While it must be conceded that literary style is in great part a matter of personal taste, it is to be regretted that a work which will come into the hands of our better educated British confrères should be thus open to criticism.

In the third place many of the 338 illustrations leave a good deal to be desired. While many of the original drawings show clearly what is intended, they are so schematic as to bear little resemblance to what is actually seen under the microscope. Moreover, many of the microphotographs are so poor that they convey but little information to the trained histologist, and consequently must be almost valueless to the average clinician. Whether this is due to the use of too thick sections, or to imperfect reproduction on the part of the publishers, it is impossible to state, although I rather incline to the latter view.

Notwithstanding these somewhat general criticisms, the work gives evidence of wide experience and great industry on the part of the author, and must inevitably serve a useful purpose in bringing before the medical public some of the problems in which all gynecologists and obstetricians should be interested.

It would lead too far to attempt to discuss in detail the scope of the work, but in general it may be said that it begins with a description of the anatomy and

histology of the normal genitalia, and ends with a chapter on the glands of internal secretion in obstetrics and gynecology while its major part is devoted to the consideration of the pathological changes which occur in the generative organs of the nonpregnant and pregnant woman.

The first section of the work is very satisfactory, while the chapter on the relation of histology to function is very interesting. I cannot agree with the author that rupture of the ripe follicle "coincides with or closely follows after menstruation," as such a view is at variance with the most recent teachings, and if correct it would imply that the orthodox Jewess should be relatively sterile instead of the most fertile of women.

The anatomical considerations in connection with prolapse of the uterus are excellent, and should be read by every one interested in the subject.

The chapter on carcinoma of the uterus contains much excellent material, and the author's advice in connection with its diagnosis cannot be too strongly emphasized—"while a specimen may be suspicious, in a given case we are dealing either with a cancer or not."

The statement that chronic oophoritis is a misnomer, and that what is usually described as such is really a small polycystic degeneration, and may even represent only a transient condition, deserves commendation and should be remembered by many who still employ the term.

To my mind the section on ovarian tumors is not particularly satisfactory, and it seems a pity to replace the time-honored term multilocular glandular cystoma by that of "pseudomucin cyst adenoma." Indeed, it seems to me that the author has missed a great opportunity by failing to describe this condition adequately, as well as the characteristic histological changes occurring in dermoid cysts and in the destruction of the tube wall in extrauterine pregnancy. Furthermore, I cannot follow his advice in the case of unilateral papillomata that the unaffected ovary should be removed prophylactically; for had I done so several families of charming children, with whom I am acquainted, would never have come into existence.

The second part of the work on obstetrical pathology is of interest, for, so far as I am aware, it represents the first attempt of the kind in the English language, outside of the scattered statements in text books on obstetrics. To the obstetrician, it is particularly gratifying to find extrauterine pregnancy classified where it really belongs; namely, under obstetrics, although I cannot agree with the implication that primary abdominal pregnancy may occur, or that rupture of a tubal pregnancy is a more frequent termination than abortion.

Many sections in this part of the work could be amplified with advantage, and with some of the statements I cannot agree: particularly when it is stated that the hepatic lesions in eclampsia are comparable to those produced by phosphorus, chloroform and bacterial poison, as it seems to me a well-established fact that the former occurs about the periphery of the lobules, while the latter commence in their central portion.

Notwithstanding such errors, the book can be strongly commended to those who are unable to use the several German works upon the subject, while the full lists of literature at the end of each section reflect great credit upon the author's learning, and serve the very important purpose of impressing the uninitiated clinician with the fact that medical knowledge is not local or even national in origin.

J. WHITRIDGE WILLIAMS.